THE OCULAR MUSCLES

HANSELL and REBER

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A PRACTICAL HANDBOOK ON THE MUSCULAR ANOMALIES OF THE EYE

BY

HOWARD F. HANSELL, A. M., M. D.

PROFESSOR OF OPHTHALMOLOGY IN THE JEFFERSON MEDICAL COLLEGE; EMERITUS PROFESSOR OF DISEASES OF THE EYE, PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; OPHTHALMOLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL; MEMBER AMERICAN OPHTHALMOLOGICAL SOCIETY; FELLOW COLLEGE OF PHYSICIANS OF PHILADELPHIA, ETC.

AND

WENDELL REBER, M. D.

PROFESSOR OF OPHTHALMOLOGY IN THE MEDICAL DEPARTMENT OF TEMPLE UNIVERSITY; PROFESSOR OF DISEASES OF THE EYE, PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; OPHTHALMOLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL; CONSULTING OPHTHALMOLOGIST TO THE RUSH HOSPITAL FOR CONSUMPTIVES; MEMBER OXFORD OPHTHALMOLOGICAL CONGRESS; PAST PRESIDENT AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

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PREFACE.

Since the authors presented their first edition of a "Handbook on the Muscle Anomalies of the Eye," the interest in this subject has grown with the increasing demands and new problems have arisen which to-day demand solution and suggestions for their practical relief.

As in the previous volume, the subject matter still represents in the main, the essence of the lectures delivered in the regular courses at the Philadelphia Polyclinic. The authors have again sought "to avoid discussions and speculations, to emphasize methods that have stood the test of their own experience, and to omit no important data that have been recognized as trustworthy."

H. F. HANSELL,
WENDELL REBER

PHILADELPHIA, PA.

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PART I.

ANATOMY AND PHYSIOLOGY.

ANATOMY.

The voluntary movements of the eye are under the control of six pairs of muscles, six in each orbit. In any and all movements the eyes are associated in their rotations and all the muscles are engaged, some actively and some passively, contracting and relaxing thus maintaining the balance or equipoise of the two eyes necessary for binocular single vision.

In the study of the functions of the muscles collectively and individually the orbital fascia must be considered (Fig. 1). Posteriorly the muscles are sheathed in delicate transparent connective tissue.

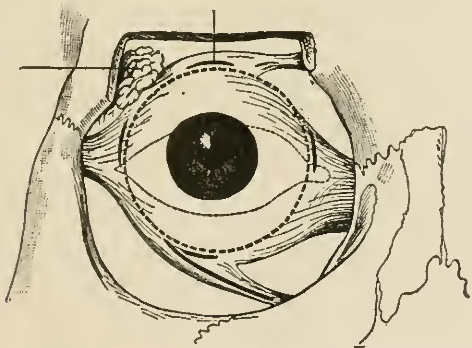


Fig. 1.—Diagrammatic representation of the relation of the globe to the orbital fascia. (Merkel and Kalius.)

When they have almost reached the eye ball the sheaths become thicker and heavier. Anteriorly the fascia divides, leaves the muscles and forms a strong fibrous sheath or cuff extending to the orbital wall where it is securely inserted. It sends off branches to support the upper division of the lacrimal gland, to unite the superior rectus with the levator palpebræ and the inferior rectus with the tarsus of the lower lid. On the nasal side the insertion of the fascia is double, the upper part forming the trochlea and

supporting the superior oblique after it leaves the trochlea; the lower is bent backward and forms in the vicinity of the posterior edge of the nasal bone a wide insertion which reaches to the floor of the orbit. The function of the fascia is to limit the effect of

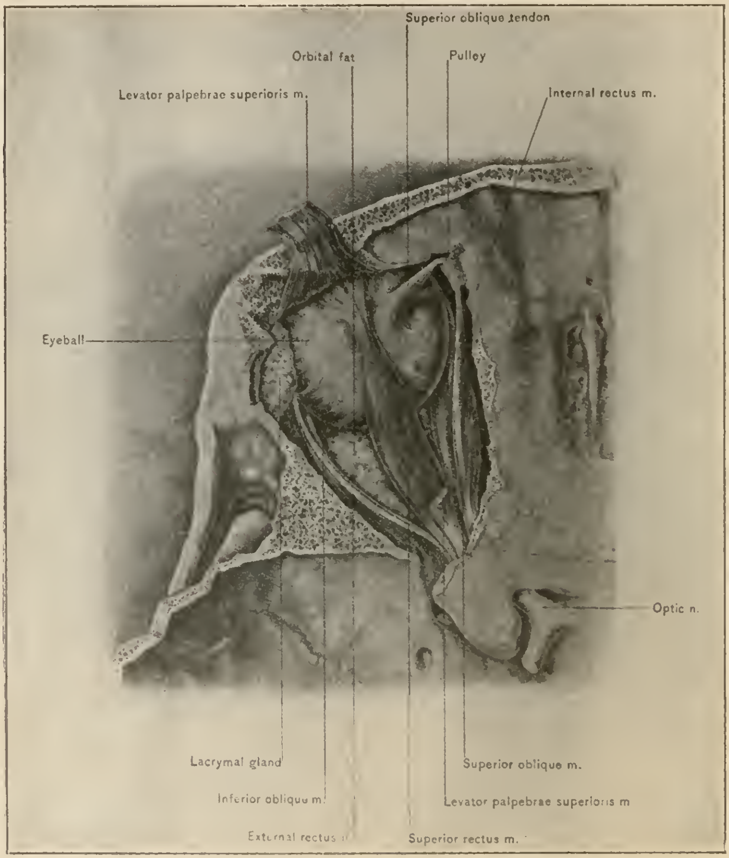


FIG. 2.—The orbital muscles. (Deaver.)

muscle action, to hold the ball firmly in its place in the orbit, and to coördinate or balance associated contraction and relaxation.

Of the six muscles in each orbit four are straight and two oblique (Fig. 2).

The **external rectus**, supplied by the 6th nerve, arises from two heads—the larger from the greater wing of the sphenoid in the external and lower border of the sphenoidal fissure; the smaller from the outer margin of the optic foramen. These two heads soon become united to form the body of the muscle which runs forward and outward, almost in contact with the outer wall for nearly half its length, and loosely bound to it by connective tissue. It is attached anteriorly to the sclera by a tendon 3.7 mm. in length, 9.2 mm. in width in a convex line, the convexity toward the cornea, 7 mm. from the outer limbus. From the tendon of the external rectus, as from those of all the other muscles, adminicula, or side attachments, are given off to the sclera which seem to strengthen the insertions. The area in cross section of its thickest portion equals 16.73 sq. mm. Its length is 45 mm. (average for adults). The external rectus controls the outer half of the field of fixation and rotates the cornea horizontally temporalward. It has no action in tilting the upper end of the vertical meriiand of the cornea either inward or outward.

The **internal rectus**, supplied by the 3d nerve, arises by a tendon common to it and to the inferior rectus from the inner margin of the optic foramen, runs forward, lying close to the inner wall of the orbit and is inserted into the sclera by an almost vertical tendon 8.8 mm. in length and 10.3 mm. in width, at a distance of 6.00 mm. from the inner limbus. The area of its thickest portion is 17.39 sq. mm. and its length is 40.8 mm. It controls the inner half of the field of fixation, rotates the cornea horizontally nasalward, and has no torsional action.

The **superior rectus**, supplied by the 3d nerve, arises by a tendon common to it and to the inferior and internal rectus at the upper margin of the optic foramen. In its course forward it perforates the orbital fat lying just under and lightly connected with the levator palpebræ, curves round the ball, pierces the layer of orbital fat and passes obliquely to its insertion into the sclera anterior to the equator and 8 mm. posterior to the corneo-scleral border, by a tendon 5.8 mm. in length and 10.6 mm. in

width. Its largest area is 11 1/3 sq. mm. and its length 41.8 mm. From these figures it will be seen that the superior rectus is the weakest of the straight muscles.

It controls the upper half of the field of fixation, turning the eye up, this vertical movement increasing as the eye is abducted and diminishing as the eye is adducted; it also rotates the cornea horizontally nasalward and tilts the upper end of the cornea in, more in adduction, less in abduction.

The **inferior rectus**, supplied by the 3d nerve, has the same origin as the internal. It lies close to the floor of the orbit and is attached to the sclera by a tendon 5.5 mm. long and 9.8 mm. wide, at a distance of 7.2 mm. from the limbus. It is indirectly, by means of the lower orbital fascia, connected with the tarsus of the lower lid. Its largest area in section is 15.85 sq. mm. and its length is 40 mm. It controls the lower half of the field of fixation turning the eye down, the vertical movement increasing with abduction, diminishing with adduction. It also turns the eye laterally in and rotates the upper end of the vertical meridian of the cornea out, these effects increasing as the eye is adducted, decreasing as it is abducted.

The **superior oblique,** a long fusiform muscle supplied by the 4th nerve, has its origin a little above the common origin of the recti from the lesser wing of the sphenoid, passing forward and upward to the inner angle of the orbit, where as a tendon it plays in, and is supported by, a fibro-cartilaginous ring in the fossa trochlearis. Upon resuming its course (the direction of which is now backward, downward and outward at an angle of 53° with the optic axis), it passes beneath the superior rectus, to be attached posteriorly to the sclera midway between the cornea and the optic nerve foramen 16 mm. from the limbus. The insertion forms nearly an antero-posterior line, the large part of the tendon being attached posterior to the vertical equator of the globe. The length of the tendon, which commences before the ring is reached and is continued to the scleral end, is 19.5 mm., and the width of the insertion varies from 6.8 mm. to 14 mm. It moves the eye down and out (temporalward) and rotates the

upper end of the vertical meridian inward, both movements increasing in abduction, diminishing in adduction.

The **inferior oblique,** supplied by the 3d nerve, arises from the anterior portion of the middle of the floor of the orbit in a depression in the superior maxillary bone just external to the anterior end of the lacrimal groove and posterior to the margin of the orbit. As a thin narrow muscle, it passes outward, backward and upward, beneath the inferior rectus, and is inserted by a horizontal attachment, about 10 mm. in width, into the sclera between the inferior and external recti on the posterior hemisphere of the ball, 17.3 mm. from the limbus. It controls the upper outer half of the field of fixation, turning the eye upward, and temporalward, which effect increases in adduction, and diminishes in abduction;[1] it also turns the eye (laterally) outward, more in abduction, less in adduction.

To Summarize.—The insertion of the internal, inferior, external and superior recti lie, in round numbers, 5, 6, 7, and 8 mm. respectively from the corneal margin, forming what is known as the spiral of insertions (see Fig. 3). Naturally, the internal rectus has therefore the greatest mechanica' advantage and the inferior rectus the next.

The **levator palpebræ,** while not strictly one of the extrinsic muscles of the ball, is closely allied, anatomically and pathologically, to them and should be included in a description of the muscles of the orbit. It arises from the under surface of the lesser wing of the sphenoid above the optic foramen, passes forward above and in close juxtaposition to the superior rectus in the first half of its course, and is inserted into the upper edge of the tarsal cartilage by an aponeurosis as broad as the cartilage. These two

[1] Volkman's results of measurement of movements, quoted by Weiland, *Archives of Ophthal.*, January, 1898, are:

Rect. Ext. moves eyeball out, up; rotates upper cornea inward.
Rect. Int. moves eyeball in, up; rotates upper cornea outward.
Rect. Sup. moves eyeball up, in; rotates upper cornea inward.
Rect. Inf. moves eyeball down, in; rotates upper cornea outward.
Obl. Sup. moves eyeball down, out; rotates upper cornea inward.
Obl. Inf. moves eyeball up, in; rotates upper cornea outward.

muscles are so intimately associated in their location and course that contraction of one involves partial contraction at least of the other.

Capsule of Tenon.—The fascias of the muscles are not so distinctly separated from each other that they may be individually described, for they are closely interwoven with themselves and with the connective tissue leaves which bind together the orbital fat.

The ball itself is surrounded by a thin, relatively strong connective tissue capsule (Tenon's) lined with endothelium which is

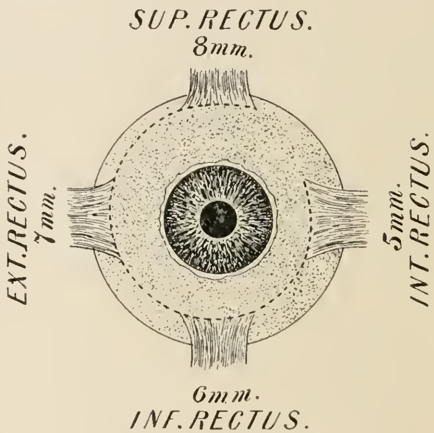


FIG. 3.—The spiral of insertions.

developed out of the sheets in the fat behind the orbit and is directly connected with them. It is connected everywhere with the ball by tender connective tissue bundles. It takes the place of the bony walls in the movement of the eyeball as in a ball and socket joint. Instead of the synovial space found in joints, the capsule of Tenon is here pierced by connective tissue bundles. It is said by Schwalbe to be a lymph space—(Tenon's space)—which is denied, however, by Langer, who says it never contains lymph nor is it connected with the lymph spaces of the eye. The "adminicula" (side attachments) of the muscles already described

are to be considered as collections of these connective-tissue bundles. Through a small opening in the capsule, the latter may be blown up easily. It ends a short distance behind the cornea, having amalgamated with the conjunctiva. Posteriorly it ends in an irregular line surrounding the ciliary nerves and blood-vessels and forming a ring 1 cm. in diameter through which the optic nerve passes. There are two layers to the capsule—one lying very close to the eyeball, the other surrounding it more or less loosely. The muscular tendons break through the capsule in slits and are connected with its edges by connective tissue.

The muscles are covered by delicate connective tissue about one-half way to the ball, then the sheath becomes heavier. This heavy fascia does not accompany the tendons through their slits in Tenon's Capsule, but bends away inward and outward. On that side toward the ball it amalgamates with the capsule and strengthens the above described strong connective-tissue ring. The outer half of the fascia becomes much thicker and gives off branches and sheaths which bend sideways and attach themselves firmly to the bone. There are three such insertions. On the upper temporal aspect the fascia from three muscles collects. The insertion is so placed that it supports the upper lacrimal gland. From it are given off the connective-tissue sheaths of the levator palpebræ and the superior rectus and the (from below) external rectus. On the nasal side the insertion is double, upper and lower, the upper to the superior oblique; the lower is bent back and forms in the vicinity of the posterior edge of the nasal bone a wide, downward inclining line which begins at the inner canthus and reaches to the floor of the orbit.

Differences of opinion prevail as to the function of Tenon's capsule. It was long believed and still is held by many authorities that the two layers of the capsule enclose a lymph space connected by lymph vessels with the suprachoroidal space, and thus form an important link in the chain of lymph spaces which connect the eye with the intracranial lymph system. Leber (Graefe Saemisch) on the other hand, asserts that it is not a lymph space, nor is it connected with the lymph spaces of the eye. Its function is

somewhat like that of a synovial membrane and permits the ocular movements without friction.

Check Ligaments.—In operations on the muscles, the prolongations of the enveloping sheaths of fascia must not be disregarded. Difficulty in picking up the tendons with the strabismus hook is often encountered, because the incision has failed to divide both layers of the capsule, and the effect desired of a tenotomy is not secured, because attention has not been given to the radical supporting attachments derived from the capsule and the check ligaments which are a portion of the orbital fascias. Serious in-

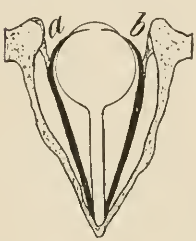


FIG. 4.—The internal check ligament (*a*) serves to prevent excessive action of the external rectus after extensive tenotomy of the internus.

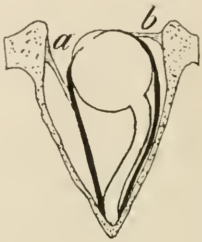


FIG. 5.—The internal check ligament (*a*) being put upon the stretch by the contracted internal rectus.

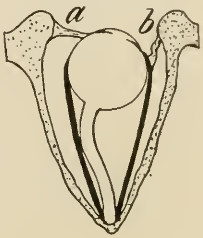


FIG. 6.—Diagram of the check ligaments. *a*, internal check ligament; *b*, external check ligament.

flammation (tenonitis), with extension of the inflammatory process backward, may result from traumatism to this serous envelope (Fig. 4).

These condensations of connective tissue which are more or less gathered together opposite the four recti muscles and are known as the check ligaments are not necessarily as sharply limited as they are shown in Fig. 4 and yet they bear a distinct relation to the neighboring muscles and they are therefore today recognized under the above name. Ordinarily no force is exerted by them, but when for instance the interni are under forced action as shown in Fig. 5 the internal check ligaments serve to prevent exaggerated action on the part of the interni muscles while the external check ligaments are relaxed. Moreover, after extensive tenotomy of

the internal rectus, the corresponding check ligament is put upon more or less constant stretch and may even limit the action of the eyeball somewhat (Fig. 6).

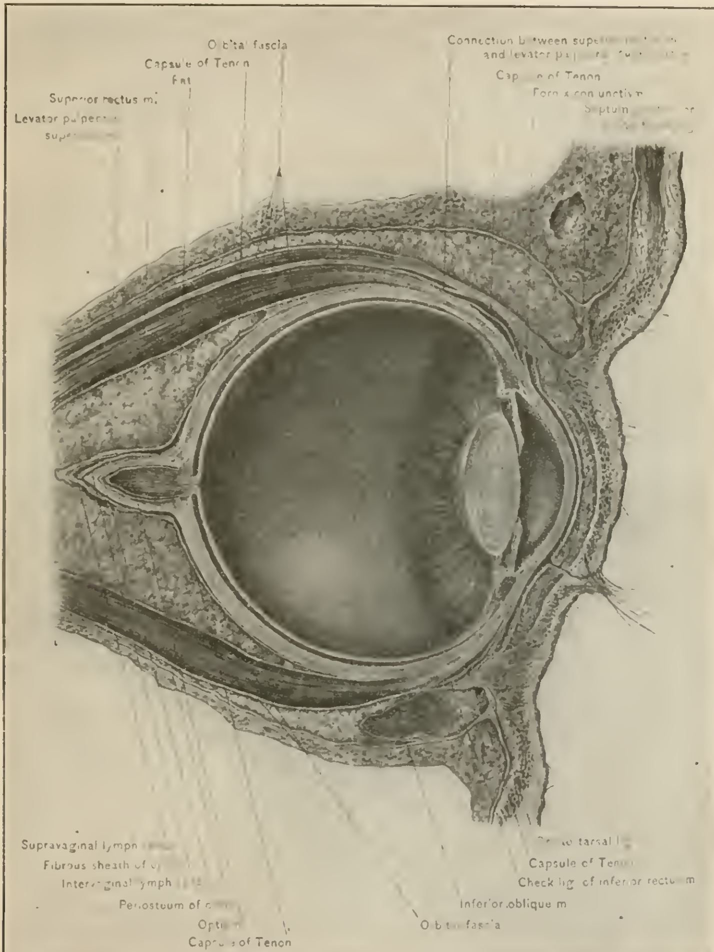


FIG. 7.—Tenon's capsule and orbital fascias. (Deaver.)

The space between the ball and the orbital walls, not occupied by muscles and nerves, is filled with fat in great part, and by con-

nective tissue. The connective tissue of the orbit is so arranged as to form light fibrous bands, probably containing elastic tissue, supporting and steadying the muscles during action and maintaining the suspension of the ball (Fig. 7).

The functions of the fascia are limitation of muscle effect; to hold the eyeball firmly in its position, to guard the conjunctiva in movements of the globe and to associate the action of the superior rectus and the upper lid, and the inferior rectus and the lower lid.

NERVE SUPPLY OF THE INDIVIDUAL MUSCLES.

The 3d, 4th, and 6th cranial nerves, known as the oculomotor, pathetic and abducens nerves, together supply the external ocular

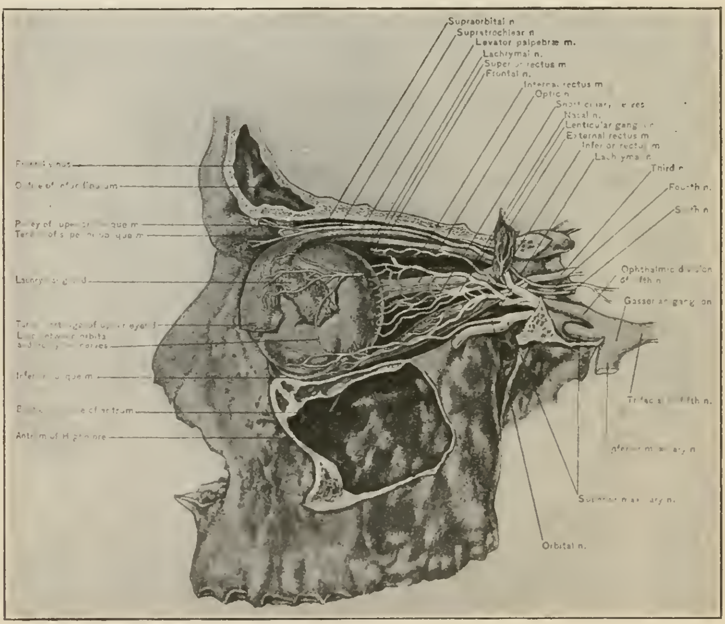


FIG. 8.—Nerves of the orbit. (Deaver.)

muscles (Fig. 8). The 3d nerve also gives off a branch to the ophthalmic or lenticular ganglion, forming its motor root to the interior ocular muscles. The scheme or hypothesis which explains the

largest number of 3d nerve nuclear problems is, probably, that of Bernheimer[1] (Fig. 9).

The vascular supply is shown in Fig. 10.

As the fibers of the third nerve leave the mass of gray substance which lays along each side of the median line, in the anterior part of the fourth ventricle, they converge as they pass downward to pierce the dura mater below the posterior clinoid process; the nerve-trunk there passes along the outer wall of the cavern-

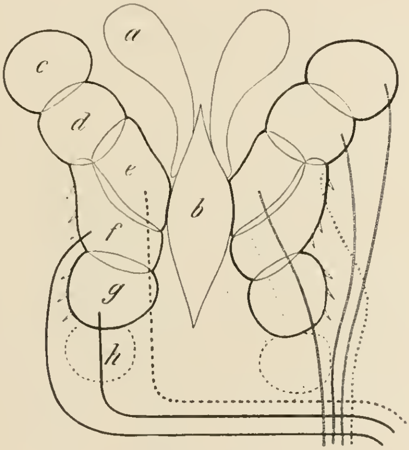


FIG. 9.—Schematic projection of the nucleus of the third and fourth nerves in the floor of the fourth ventricle. *a* and *b*, nucleoli for the intraocular muscles (iris and ciliary muscle); *c*, levator nucleolus; *d*, superior rectus nucleolus; *e*, internal rectus nucleolus; *f*, inferior oblique nucleolus; *g*, inferior rectus nucleolus; *h*, fourth nerve nucleus or superior oblique nucleus. (Bernheimer, Graefe-Saemisch Handbuch, second edition.)

ous sinus, enters the orbit through the sphenoid fissure and divides into two branches. The superior division supplies the superior rectus and levator palpebræ; the inferior division separates into three branches, one going to the internal rectus; and the third, and largest, to the inferior oblique; and one to the inferior rectus. A fourth goes to the short root of the lenticular ganglion. From the lenticular ganglion are given off the long ciliary nerves which

[1] Some authorities prefer Perlia's: others Kahler & Pick.

perforate the sclera around the optic nerve, pass forward between the sclera and choroid and are distributed to the iris and ciliary muscle. The terminal branches anastomose finally with the terminals of the seventh. The nerve-trunk also receives filaments from the cavernous plexus of the sympathetic, and its superior division is not infrequently connected with the ganglionic branch of the nasal nerve.

FIG. 10.—Vascular supply to the oculomotor nuclei. *a*, Roof of the corpora quadrigemina; *b*, acqueduct of Sylvius; *c*, sphincter-iridis and ciliary muscle nucleus; *d*, *f*, and *g*, vascular supply to the remaining nuclei. (Bernheimer, Graefe-Saemisch Handbuch, second edition.)

The 4th, pathetic or trochlear nerve supplies the superior oblique muscle. Its deep origin can be traced to the nucleus which lies beneath the aqueduct of Sylvius, behind and on a lower level than the third nerve nucleus. The fibers pass as they emerge to the dorsal aspect of the aqueduct and decussate with the fibers of the opposite side?). The nerve-trunk pierces the dura mater near the posterior clinoid process, passes with the 3d nerve along the outer wall of the cavernous sinus and enters

the orbit through the sphenoidal fissure, being the highest of the nerves passing through the orbit. It also receives filaments from the cavernous plexus of the sympathetic, and transmits a twig to the lacrimal.

The nucleus of the 6th or abducens nerve consists of multiple ganglion cells lying immediately under the center of the floor of the 4th ventricle on either side of the median sulcus. It is bordered on its inferior, inner and upper sides by the first, second and third portions of the facial nerve. The fibers run obliquely downward to emerge at the lower border of the pons. The connections of the 6th nerve nucleus are said by Bruce (*loc. cit.*) to be as follows: with the second part of the root of the facial nerve and the segment of the 3d nerve nucleus supplying the internal rectus; with the auditory nucleus and with the cortex of the opposite cerebral hemisphere, and perhaps with that of the same side. The nerve penetrates the dura mater on the basilar surface of the sphenoid bone, passes through the posterior or clinoid processes, enters the cavernous sinus, and finally the orbit through the sphenoidal fissure, to be distributed to the external rectus muscle from its inner surface. It also receives filaments from the carotid and cavernous plexus of the sympathetic nerve, from Meckel's ganglion and from the ophthalmic nerve.

VASCULAR SUPPLY.

The vascular supply to the muscles is derived from the muscular branches of the ophthalmic artery, one of the terminal branches of the internal carotid.

The ophthalmic artery at its origin is 2 mm. in diameter. It passes through the optic foramen under the optic nerve, supplying its sheaths and perineurium, later perforating and supplying the nerve-substance itself. Soon after its entrance into the orbit, it gives off the muscular branches. All the blood-vessels of the orbit are characterized by their tortuous course and their lax attachments to flexible structures. They are thus enabled to accompany the movements of the ball without tearing.

The muscular arteries are two in number, the smaller passing

up and medianward, the larger downward and laterally, supplying the muscles in their course. They give off the anterior ciliary branches which perforate the tendons of the muscles and the sclera, and supply the ciliary region and iris with blood. (Section of one of these arteries in tenotomy often gives rise to profuse, but never alarming hemorrhage.)

The arteries are accompanied by veins situated similarly and described under similar names.

The most important orbital vein is the superior ophthalmic, with which all venous branches in the orbit are in direct or indirect connection. It collects blood from the frontal, nasal, supra-orbital, angular, venæ vorticosæ, and the central retinal veins. Its course is along the superior rectus, backward in the muscular cone, emptying into the cavernous sinus.

PHYSIOLOGY.

INNERVATION.

The muscles governing the movements of the eyeball, the accommodation and the iris are innervated by the 3d, 4th and 6th pair of cranial nerves and the carotid plexus of the sympathetic system. The former have their deep origin in the posterior extremity of the aqueduct of Sylvius and the anterior part of the floor of the 4th ventricle. The muscular movements are voluntary because they are represented by distinct areas in the cortex. From these cortical centers are derived nerve fibers which run indirectly to the nuclei and undoubtedly have connections with other centers in the brain, the functions of which are associated. The fibers have not been dissected or strictly outlined. Their presence must be assumed in the explanation of mental processes, part of the evidence of which are the voluntary, although not always conscious, ocular movements. The nuclei on the other hand have been studied, their exact location, their relations to each other and their functions to a large extent determined. From the nuclei large numbers of nerve fibers are given off which immediately unite to become distinct nerve trunks easily seen at the base of the brain and followed to their exit through the sphenoidal fissure to be distributed to their respective terminations in the muscular tissues and probably anastomosing with the terminal branches of the 7th nerve.

Russell's experiments have led him to conclude that the cerebellar cortex plays no little part in ocular movements and that it is associated with the cerebrum in these functions. The accompanying drawing, Fig. 11, based on Russell's experiments, indicate the areas which are supposed to preside over the different eye movements. It will be observed that they are above the center of the fissure of Sylvius just anterior to the large motor

area, and, moreover, that they are close to the facial area, and on the left side.

NUCLEAR CENTERS.

The mass of cells composing the nucleus of the 3d lies on both sides of the median line next to the corpora quadrigemina and under the aqueduct of Sylvius anterior to and on the floor of the 4th ventricle. The nucleus is from 6 to 10 mm. in length and of varying breadth mingling imperceptibly with adjacent cells. Posteriorly they encroach upon the cells of the 4th

FIG. 11.—The external surface of the right half of the brain of the ape (Macacus Sinicus). (After Russell, *Journal of Physiology*, vol. 17, p. 8.) 1, Upward movement of both eyes; 2, downward movement of both eyes; 3, movement of both eyes upward and to the opposite side; 4, movement of both eyes downward and to the opposite side; 5, convergence. The numbers denote the chief foci, excitation of which evoked movements of the eyes other than conjugate turning to the opposite side from the hemisphere stimulated.

nucleus without a distinct line of demarkation between them. The mass may be divided into nucleoli each with its separate function and muscle control. In the accompanying diagram the anatomy and physiology is schematically shown.

It will be noticed that from some of the nuclei the fibers run direct to the muscles on the same side, while others are crossed to stimulate those on the other side, the crossing taking place mostly in the anterior half of the nucleus. Speaking generally the inner or median part of the nucleus belongs to the intraocular muscles and the outer portions to the extra-ocular muscles. The

anterior and principal part of the nuclear masses belongs to the 3d nerve. Adjoining the proximal or posterior end of the posterior corpora quadrigemina on both sides are the nuclei, right and left, of the trochlearis. Much farther back and in juxtaposition with the facial muscles and fibers is the nucleus of the abducens.

CORTICAL CENTERS.

Having conducted a large number of experiments on monkeys to determine the site of the centers for eye movements, St. Bernheimer (Graefe Saemisch, second edition) has reached the following conclusions: 1. The gyrus angularis and especially its middle part of both hemispheres is the only cortical center for synergic eye movements. 2. The right gyrus angularis controls movements toward the left, up and left, and down, the left gyrus controls movements toward the right, up and right, and down. 3. The anterior corpora quadrigemina are neither a reflex center for eye movements nor the passage for the neurons. 4. Since after median section of the brain between the aqueduct of Sylvius and the nuclei region of the eye muscles irritation of both angular gyri produces no eye movement, therefore the connection-neurons between the nuclei and the cortex are all crossed in the angular gyrus in the median line under the plane of the aqueduct of Sylvius between it and the nuclei. 5. The end filaments of the connecting fibers communicate probably by other cells (schalzellen) with the roots of the motor ganglion cells of the nuclei. 6. The schalzellen lie probably imbedded and scattered in the central gray matter and form no cell mass. 7. In consequence of partial crossing of the 3d, the total crossing of the 4th, and the connection of all the oculomotor nuclei with each other, it may well be asked whether the crossing connecting fibers of one angular gyrus equally influence the muscles of both eyes?

THEORY OF INNERVATION.

To outline the structure and function of each of the individual eye-muscles is a relatively simple matter, yet it is by no means so

easy a task to give a satisfactory explanation of the innervational state of the eye-muscles corresponding to all the positions the eyes may assume in monocular or binocular fixation. The theory that has gained the widest acceptance is the so-called innervational theory, so ably championed by Hansen Grut, 1898. It provides that every contraction of a muscle or set of muscles to produce ocular movement in a given direction is accompanied by relaxation of the muscle or set of muscles that are directly antagonistic to the contracting muscles, which relaxation keeps even pace with the contraction going on in the muscles producing the deviation. As the eyes are capable of being easily rotated there must, in order that single vision be preserved, be a balance among the forces that tend to move the eyes in various directions. This balance is possible only under two conditions: 1, that all the muscles be slack and exert no tension (an untenable hypothesis); or 2, that all the muscles so tend to contract as to cause an equal rotational strain in all directions. Hence, we suppose that the muscles are always in a state of partial tonic contraction, at any rate during the period of full consciousness.

There is no limitation to the directions in which the eye can be rotated by the action of one or more muscles, and under certain circumstances a contraction of all the muscles produces a slight enophthalmus.

The primary position of the eyes is observed when an individual holds the head erect and gazes straight in front toward infinity in the horizontal plane of the eyes.

The predominately active muscles in the following movements from the primary position are:

Right and left turning; only the rectus externus and rectus internus.
Upward turning; rectus superior and inferior oblique.
Downward turning; rectus inferior and superior oblique.
Up and out turning; rectus superior, inferior oblique, and rectus externus.
Down and out turning; rectus externus, rectus inferior and superior oblique.
Down and in turning; rectus internus, rectus inferior, and superior oblique.
Up and in turning; rectus internus, rectus superior and superior oblique.

These are the eight principal secondary positions, and in all the degrees of the circle between them the rotation will be accom-

plished by the combined action of the muscles normally turning the cornea to these positions. The extent of the turnings in the field of fixation varies, according to different observers. The average is probably as follows:

up	up-out	up-in	out	in	down	down-out	down-in	
40–50	35–50	40–55	35–55	40–48	40–70	35–60	25–60	(*Duane*).

The discrepancies arise from variations in the normal power of the muscles of one individual as compared with another, and the degree of attention and effort of which an individual is capable. Also, in determining the rotation by Stevens's tropometer, the form of the ball must be taken into consideration. Such wide variations, as the averages given above, are confusing to one who endeavors to determine whether a certain movement is normal or otherwise.

GENERAL PHYSIOLOGIC CONSIDERATIONS.

The Law of Projection.—To properly understand the phenomena of vision, certain peculiar functions of the retina, optic nerve, and associated brain apparatus must be clearly apprehended, for they lie at the very basis of these phenomena.

While it is true that images are formed on the retina, we do not actually see the retinal images. In fact, we do not actually see anything *in the eye* but something *outside in space.* The retinal image is conveyed by way of the optic nerves and tracts to the brain, there to determine certain changes. These *changes* or *effects* the brain then refers or projects *outward* in a definite direction into space as an external image or facsimile of the object which produces it. So that what we see as external images are really projections outward of retinal images. This law of outward projection is important. It is not a new law, specially made for the sense of sight, but only a modification of a general law of sensation. This general law is that irritation or stimulation in any portion of a sensory fiber is referred to its peripheral extremity. If the ulnar nerve is pinched in the hollow on the inner side of the point of the elbow, pain is felt in the little and ring

fingers, where this nerve is distributed. In the case of the optic nerve the impression is so wholly projected outward that we are unconscious of any sensation in the eye at all. Hence what we are accustomed to call the field of view is nothing else than the external projection into space of various retinal stimulations.

The Law of Direction.—The direction of external projection may be exactly (or nearly exactly) defined as follows:

The central ray of each point in the field of view passes straight through the nodal point of the lens without deviation to the retina.

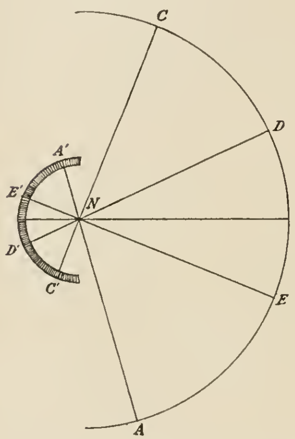


FIG. 12.—Scheme of the law of direction. The larger circle represents the limits of space; the smaller one the retina.

It is evident then that every retinal rod and cone has its inevitable related point or spot in the field of view (visual field) and vice versa. The two points—retinal and spatial—exchange with one another by impression and outward projection along the straight lines connecting them. This is represented in Fig. 12 in which S S represents the spatial concave and R R the retinal expanse, with straight lines of rays of light connecting. A ray from a point *c* in space passes in a straight line through the nodal point of the lens *n*, and strikes a certain retinal rod *c*; that impression is projected by the rod (or referred by the brain) back along the ray line to the place whence it came. Study of the figure shows that the position of all retinal images must be the reverse of the objects in space—that the upper part of the field of view corresponds to the lower part of the retina and the lower part of the field of view (visual field) to the upper part of the retina. Similarly the right and left sides of the visual field are related to the left and right sides respectively of the retina.

These two laws—the law of external projection and the law of direction—are two of the most fundamental laws of vision.

The first shows why objects are seen externally in space; the second gives the exact place where they are seen, that is to say where the brain locates them. The general law of projection is not followed very exactly with respect to the very forwardmost portions of the retina in the region of the ora serrata.

What has been thus far said treats only of the phenomena of monocular vision. But most individuals possess two eyes, and these are not to be considered as mere duplicates so that if we lose one we still have another. Quite on the contrary, the two eyes ordinarily act together as one instrument. There are many visual phenomena and many judgments based upon these phenomena which result entirely from the use of two eyes as one instrument. The phenomena of binocular vision are far less purely physical than those of monocular vision. They are also more obscure, illusory and difficult of analysis because more subjective and more closely allied to psychical phenomena.

Corresponding or Identical Points.

As is well known, the retinas are two deeply cup-shaped expansions of the optic nerve. R and L (Fig. 13) represent a flat (or Mercator) projection of these two cups. The black spots in

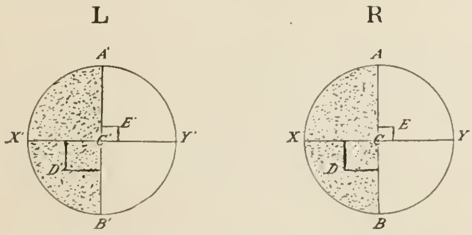


FIG. 13.—Mercator projection of the two retinas; showing corresponding retinal points.

the centers represent the central spots. If now we draw vertical lines (vertical meridians) A, B, A′, B′, through the central spots and divide the retinas into equal halves, then the left or shaded halves would correspond point for point; i.e., the internal or nasal

half of one retina corresponds with the external or temporal half of the other and vice versa. More accurately if the concave retinas be divided by coördinates like the lines of latitude and longitude of a globe (ab, and xy being the meridian and equator), then points of similar longitude and latitude in the two retinas, as D,D′, and E,E,′ are corresponding points. Of course the central spots will be corresponding points; also points on the vertical meridians, A,A′, B,B′, at equal distances from the central spots will also correspond.[1] These phenomena therefore afford the basis for the following:

Law of Corresponding or Identical Retinal Points.—*Objects are seen single when their retinal images fall on corresponding or identical retinal points.*

Of course it is true that as there are two retinas there are naturally two retinal images of every external object, and since, as has been shown, retinal images are projected outward into space as external images, one will certainly have two external images of every object. If then there are two external images for every object, it may be asked why are not all objects seen double? And it may be answered that all objects are indeed seen double except under certain conditions.

PHYSIOLOGIC DIPLOPIA.

The phenomena of double images of all objects except under certain conditions is a fundamental one in binocular vision, yet it is commonly overlooked by even the most intelligent persons unaccustomed to analyzing their visual impressions. If one holds up one's index finger at arm's length and then looks not at the finger, but at the wall or the ceiling or the sky or any sufficiently removed expanse, two images of the finger will be seen, the left one belonging to the right eye and the right one to the left eye. This is easily proven by shutting first one eye and then the

[1] In some eyes the apparent vertical meridian which divides the retinas into corresponding halves is not perfectly vertical, but slightly inclined outward at the top. This would effect all the meridians slightly, but the effect is insignificant.

other and observing which image disappears.[1] To confirm this phenomenon another experiment may be tried. With the two forefingers placed directly in front of one, in the median plane of the head one at arms length, the other one half that distance (see Fig. 14) it will be observed that if the farther finger is seen single, the nearer one is seen double; if the nearer finger is ob-

FIG. 14.—Experiment to illustrate physiologic diplopia. If the far finger is looked at the near finger is seen double (crossed diplopia); if the near finger is looked at, the far finger is seen double (uncrossed diplopia).

served this one will be seen single, but the farther one double; when the farther one is seen double, the *right* image disappears when the *right* eye is closed; that is to say the images are *uncrossed* or *homonymous.* When the nearer one is seen double, the *right* image disappears when the *left* eye is closed; that is to say the images are *crossed* or *heteronymous.* The important fact to be borne in mind is that it is impossible for both fingers to be seen as single objects at the same time. Therefore it is evident that when we look directly at anything we see it single, but that all

[1] Some persons find difficulty in consciously recognizing the two images. It is not uncommon for such persons to habitually neglect one image until it finally drops out of consciousness. Hence they are likely to become either right or left eyed.

things situated at that same instant either nearer or farther away than the object looked at are seen double whether consciously recognized as double or not. Fortunately, in ordinary every day use of the eyes this physiologic diplopia is disregarded because of the overwhelming dominance of the fusion sense and single vision results.

ON SYNERGISTIC MUSCLES, OR THOSE WHICH ASSIST EACH OTHER.

It has been shown in the chapter on anatomy and physiology that the external rectus and the internal rectus have purely lateral action; but that the superior rectus moves the eyeball not only upward, but also inward (medianward) while the inferior rectus draws the eyeball downward and also inward (medianward). Also, that the superior oblique muscle moves the cornea

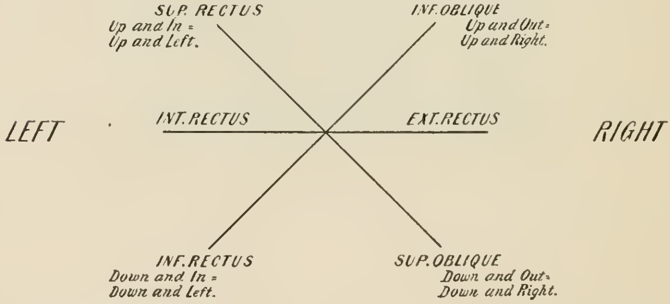


FIG. 15.—Showing the dominant action of the muscles of the right eye. This diagram is based on the presumption that the eyes are in the primary position when the movement in the various directions begins.

downward and outward (temporalward) and the inferior oblique moves the eyeball upward and outward (temporalward). These movements may be represented diagramatically as in Fig. 15. As a matter of convenience and memory help the student will do well to remember that the right inferior oblique for instance moves the right eyeball up and right rather than up and out or temporalward. The same is true of all other motions imparted by the muscles to the globe.

Further study of Fig. 15 (which refers to the right eye) will show that both the superior rectus and the inferior oblique of the right eye are elevators of that eye and that when they act together they rotate the eyeball directly upward, their torsional actions in opposite directions neutralizing each other. These two muscles are therefore said to be synergistic muscles. Similarly the superior oblique and the inferior rectus of the right eye are both depressors of the right globe (their torsional effect being neutralized) and they also are synergistic muscles.[1]

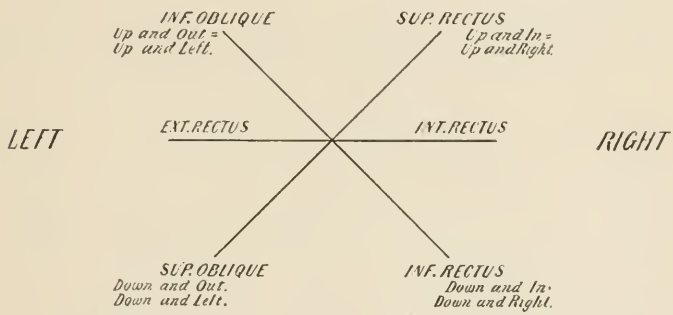


FIG. 16.—Dominant action of the individual muscles of the left eye.

In considering Fig. 16 which refers to the left eye, it will be found that exactly the same state of affairs obtains so far as the left eye is individually concerned, only it will be seen at a glance that while the right superior rectus moves the right globe up and left, the left superior rectus moves the left globe up and right. The same difference obtains with the inferior rectus of the two eyes and with both obliques, and these differences must be closely observed as they lead directly to the study of the eyes as a pair.

[1] It can also be easily seen that in moving the right eyeball directly to the right the external rectu predominates in that movement but the superior and inferior oblique by joint contraction assist in that movement; also that in the movement of the right eyeball to the left, the internal rectus dominates the action but is assisted by the joint contraction of the superior and inferior rectus. This synergism obtains in every eye movement that is made and there is no movement of the eyeballs in which but one muscle is the sole and only muscle concerned.

ASSOCIATED MUSCLES.

If Figs. 15 and 16 are now studied side by side it will be seen at once that in the movement of both eyes directly to the right, the muscles that predominate in this action are the right external rectus and the left internal rectus. In movements to the direct left, the right internal rectus and the left external rectus dominate the movement. In movement of the eyes up and to the right the two muscles principally concerned are the right inferior oblique

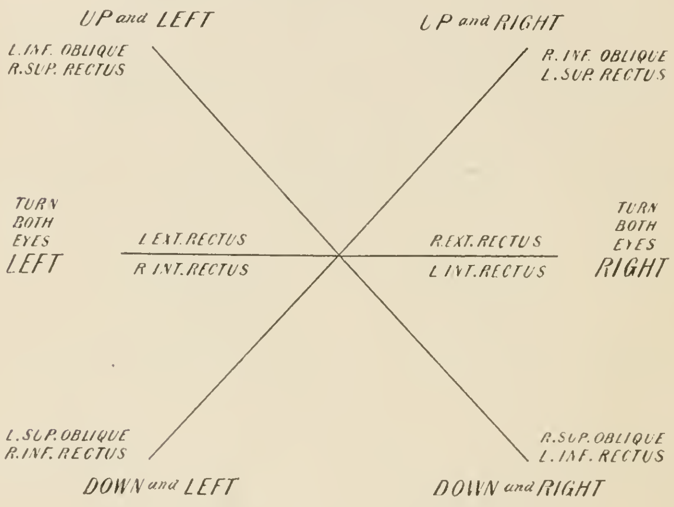


FIG. 17.—Representing Figs. 15 and 16 superimposed. The associated muscles in the two eyes are thus graphically shown.

and the left superior rectus; down and to the left, the right inferior rectus and the left superior oblique, and so on through the six principal movements of the eyes. In each one of these six principal movements of the eyes two muscles are primarily active —one in each eye. This yoking of one muscle in one eye with another in the fellow eye to perfect the ease of binocular movements is known as association of the muscles. Hence such muscles are designated *associated muscles* (or yoke fellows). Figs. 15 and 16 may now be superimposed to produce (Fig. 17) a diagram representing perfectly the associated muscles in both eyes. It shows that

in movements of the eyeballs up and to the right, the right inferior oblique and the left superior rectus are the dominant forces; in movements down and to the left, the right inferior rectus and

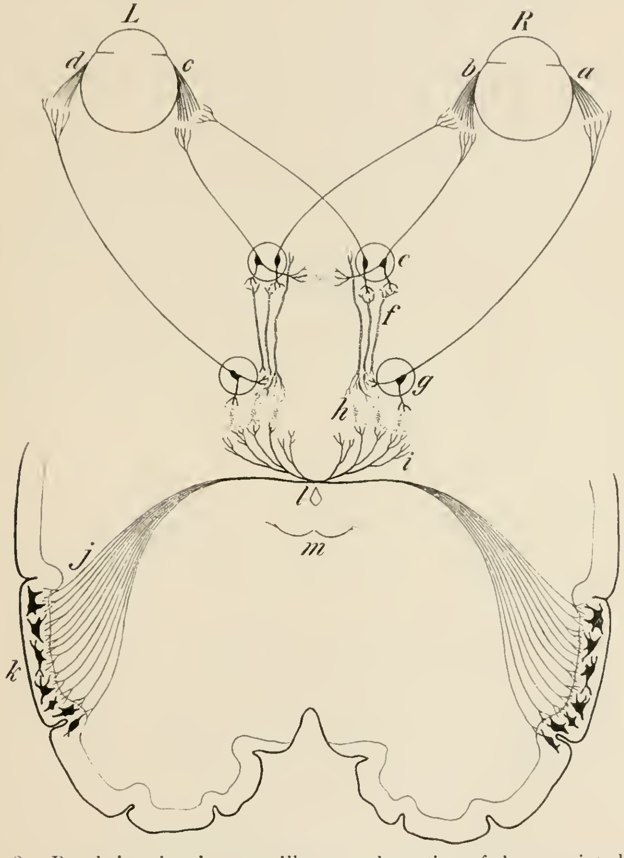


Fig. 18.—Bernheimer's scheme to illustrate the action of the associated muscles in lateral movements (lateral conjugation) and in convergence. *a*, Right external rectus; *b*, right internal rectus; *c*, left internal rectus; *d*, left external rectus; *e*, third nerve nucleus; *f*, communicating fibres; *g*, sixth nerve nucleus; *h*, fimbriated cells; *i*, arborizing ends of the fibres to the opposite side of the cerebral cortex; *k*, cortical centers; *l*, aqueduct of Sylvius; *m*, roof of he corpora quadrigemina. (Graefe-Saemisch Handbuch, second edition.)

the left superior oblique do most of the work and so on through all the six cardinal movements imparted by the muscles to the eyes. In each of these movements as shown on the diagram,

the muscles shown there as associated, work most harmoniously together. These associated movements are also known as conjugate movements.

From what has thus far been said, it naturally follows that the two eyes work together as one, as Hering has said. It is impossible for one eye to move in one direction without the other one paralleling its action in every particular. A nervous impulse from the cortex must of necessity be divided equally between the two eyes. Hence the perfectness of the conjugation between them. These association or conjugate movements of the eyes are represented in all likelihood by centers in the anterior central as well as the posterior cortex. Sherrington's, and Risien Russell's experiments both strongly support this idea[1] (Fig. 18).

Five conjugate innervations have long been known, namely:

1. Binocular elevation.
2. Binocular depression.
3. Binocular right rotation.
4. Binocular left rotation.
5. Convergence. A distinct act.

There are probably a number of others, but they remain at present speculative.

ANTAGONISTIC MUSCLES.

In addition to synergistic and associated action of the muscles, it is well to keep in mind always the antagonistic action as set forth in the following table:

Muscle.	*Antagonists.*
Internal rectus.	1. External rectus.
	2. Superior oblique.
	2. Inferior oblique.
External rectus.	1. Internal rectus.
	2. Superior rectus.
	2. Inferior rectus.

[1] There may be sub-cortical stations for additional control of such movements, but of such connection the seeming proof is much less probable.

Superior rectus.	1. Inferior rectus.
	2. Superior oblique.
Inferior rectus.	1. Superior rectus.
	2. Inferior oblique.
Superior oblique.	1. Inferior oblique.
	2. Superior rectus.
Inferior oblique.	1. Superior oblique.
	2. Inferior rectus.

The figures 1 and 2 signify primary and secondary antagonists respectively.

THE STEREOSCOPE.

The most important information on the distance of an object is furnished us by the degree of convergence necessary to fix it

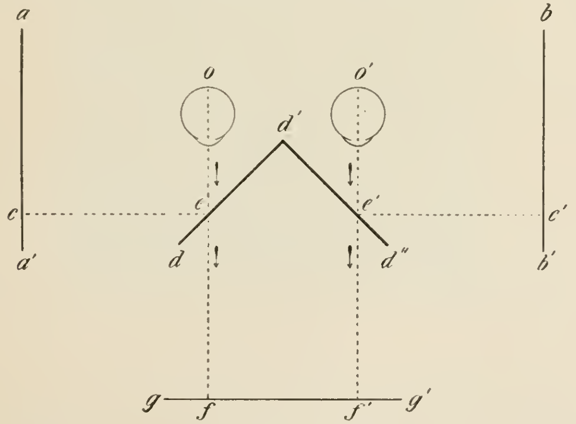


FIG. 19.—Wheatstone's stereoscope, composed of two plane mirrors *d*, *d′* and *d″* which are at right angles to each other. The eye *O′* sees in the mirror *d′d″* the picture *bb′* and projects it to *ff′*. The eye *O* sees in the mirror *dd′* the picture *aa′* and projects it to *ff′* just as the eye *O′* does; the two images are fused into a single one, presenting the phenomenon of relief. In order not to have the relief reversed (or pseudoscopic) it is necessary to present to the left eye the image intended for the right eye since the mirrors reverse the images.

binocularly. When we fix a distant object, a near object appears in double crossed images. Although these double images are not often perceived, they give us, nevertheless, a vague idea of the

distance of the object, for they furnish a pretty accurate impulse to convergence, and thus guided we converge for the object without much effort. So that two visual judgments have been formed—one for the remote object and one for the nearer object, and the difference between the two judgments furnishes the information on which we base our estimate of the distance of objects. When we look at an object having considerable depth or space, or at a scene, there is an image of the object or scene formed on each retina. These two images are not exactly alike because they are taken from different points of view. If two cameras are placed before an object or a scene with a distance between them of 2 or 3 feet, they will fairly well present two great eyes, with large interocular distance. The pictures thus taken cannot be exactly alike because taken from different points. They will differ from each other exactly as the two retinal images of the same object differ, only in greater degree. If these two photographs be binocularly combined in a stereoscope they ought to and must produce a visual effect exactly like an actual object or scene, for in looking at an object or scene we are only combining retinal images exactly like these pictures, because they are taken in the same way. The advantage of binocular vision was made clear only by the invention of the stereoscope of Wheatstone in 1833 (Fig. 19). With this instrument we obtain an impression of depth much superior to that which any other representation can give of it. The stereoscope facilitates binocular combinations beyond the plane of the pictures. The refracting stereoscopes (Brewster's, 1849, Fig. 20) by means of lenses, supplement the lenses of the eyes and thus produce on the retinas perfect images of a

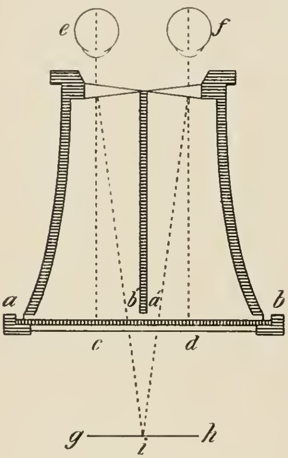


FIG. 20.—Brewster's stereoscope. The pictures *c* and *d* are blended or fused in the plane *gh*.

near object, although the eyes are looking at a distant object. The lenses also enlarge the images and thus complete the illusion of a natural scene or object. The clinical value of the refracting stereoscope is indicated by the number of modifications that it has undergone. The Holmes model (Fig. 21) is the one ordinarily found in the shops. Javal, Parinaud, George Bull, Richard Derby, Oliver and many others have all brought forward various devices to adapt the stereoscope to every day ophthalmic practice, each with its own peculiar excellences. The one disadvantage of

FIG. 21.—Holmes' stereoscope.

all of them was that they were very imperfectly, if at all, adaptable to the higher degrees of deviation of the ocular axes (heterophoria and heterotropia). Javal was the only one who met this difficulty with his hinged stereoscope with mirrors, concerning which he writes:

"In spite of the care that has been taken to increase the field of the stereoscope by the employment of lenses of relatively short foci, it often happens that the deviation is too strong to enable one to use a stereoscope which has lenses at all. It is then that one finds the advantage of employing my *stereoscope-a-charniére*."

About ten years ago Worth carried the idea further. He put two of Priestly Smith's fusion tubes on a hinge and bending each tube he placed a mirror at the bend, making an adjustable reflecting stereoscope. It has one advantage over Javal's in that the brightness of the images may be varied for either eye—a point of value in the orthoptic treatment of strabismus.

THE EVOLUTION OF BINOCULAR VISION.

Binocular single vision is the normal state of human eyes. This is because of the partial decussation of the optic nerve fibers in the chiasm. When compared with the visual apparatus in the lower animals the study of binocular vision takes on great interest. The gradual evolution of the invertebrate eye is scientifically satisfactory. The transition from the invertebrate to the vertebrate eye is involved in some doubt. From that point upward, however, the evolution is resumed and continues very regularly. In the lowest class of vertebrates (the fishes) the eye is probably no better than a squid's. Their eyes are situated on the side of the head with such widely divergent axes that there is no overlapping of the visual fields at all. There is no consensual movement, each eye being absolutely independent of the other. There is no common field of view, no common point of sight, no corresponding points in the two retinas; hence no binocular single vision. Leaving out amphibians and reptiles (of which we know little), in birds binocular vision becomes possible by an unique arrangement of corresponding points about a very excentral fovea, though their optic axes are widely divergent. In mammals the optic axes are brought around more and more to the front and the convergence of the axes on a point of sight at infinity becomes easier and easier. With this comes the gradual development of corresponding points about a more highly organized central area on which is based all the phenomena of binocular vision and the visual judgments issuing therefrom. Only in the anthropoid apes do we find the eyes fairly in front with optic axes parallel in the position of rest. There is too at

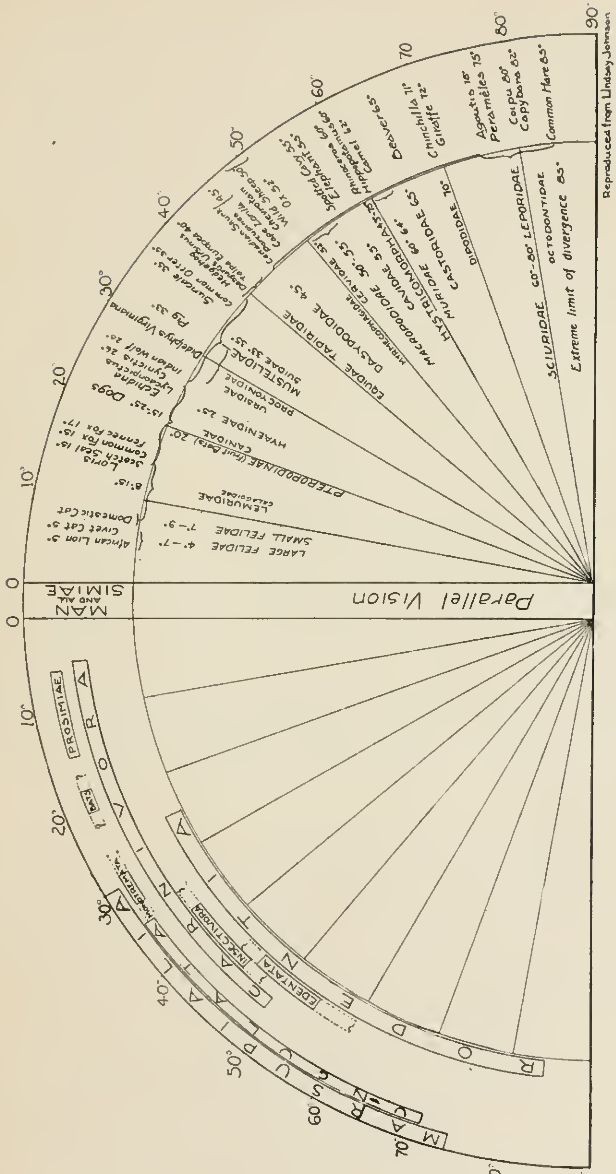


FIG. 22.—Lindsay Johnston. Scheme of the visual axes in the mammalia.

this stage a much more highly organized spot of central vision (fovea) added, which is one of the essentials in what the psychologist calls the faculty of attention. The existence of the fovea is necessary to the concentration of attention on the thing looked at, for "how could we attend to one thing if all other things were equally distinctly seen?" The work of Lindsay Johnson[1] has added much certainty to this study. Each time he investigated the fundus oculi of the more than 200 animals that came under his observation, he measured the divergence of the optic axes by means of a special contrivance known as a goniometer. The results of this tremendous task are shown in Fig. 22. On the left hand of the chart will be found the range of divergence between the various members of the orders taken as a whole, while on the right, details as to families and genera are given. It will be readily seen from the chart that the higher the order the nearer the axes approach parallel vision, although the range in each is considerable, and each one to some extent overlaps the other. It will also be noted on the right hand that the degree of divergence for the hare is 85 degrees, for the giraffe 72 degrees, for the porcupine 45 degrees, for the hedgehog 40 degrees, for the pig 33 degrees, for the wolf 25 degrees, for the dog 20 degrees, for the fox 15 degrees, for the cat 10 degrees, and for the diurnal monkeys and apes, as also in men, zero, or parallelism of the optic axes.

In the development of the human embryo there can be followed the same evolution in one species that is above outlined in all the vertebrate. The eyes develop on the sides of the head as in the fish. Gradually with the formation of the face they are brought around to their permanent frontal position and are in place about the end of the second month. In this the ontogeny agrees with the philogeny and the forward rotation of the optic axes of each vertebrate is consistently followed in the developing human enbryo.

The stimulus to the animal to see what is in front of it with

[1] Taken from his work, "The Comparative Anatomy of the Mammalian Eye," London, 1901.

both foveas directed exactly to the one point, attains its highest development in the anthropoid apes and man.

In the newborn human infant there is no coördination of the eyes. After the first week of life the babe will sometimes fix a small electric light or candle momentarily, but only momentarily. Then the eyes will wander aimlessly about, being sometimes in convergence and sometimes in divergence. By the end of the third week this fixation will be a little more fixed and by the time the sixth to the eighth week is reached many babes will fix the small electric light in a dark room and maintain the fixation for quite a time. The nurse or mother will state at this time that the child is "beginning to take notice of things." This is what the psychologist terms "the unfolding of the faculty of attention." By others it is known as the fusion faculty. This faculty is not firmly established before the end of the sixth month and occasionally not before the end of the first year, and even then slight illnesses (especially gastro-intestinal ones) may temporarily disturb it gravely. It is a faculty that in some children responds rapidly to training as late as the sixth or seventh year, but very poorly after that period.

Binocular single vision therefore may be defined as a *congenitally acquired psychic blending of the two sets of visual impressions that are focussed on corresponding positions of each retina.*

TESTS FOR BINOCULAR VISION.

Of all the tests for binocular vision or perception of relief, Hering's drop test is probably the most reliable, for in most others we have to rely upon the patient's statements without being able to verify them. A crude model (Fig. 23) can easily be made by taking a flat pasteboard box about 1 foot long, 2 inches high and 6 to 7 inches wide, made concave at one end to fit tightly against the head. From the other end two wires project forward and out-

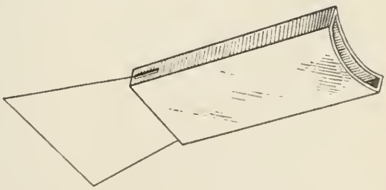


FIG. 23.—Hering's binocular vision test box.

ward connected at their ends by a horizontal thread which is provided with a small bead at its midpoint for the patient to look at through the box. Small objects, such as dried peas or beans or small marbles are dropped by the examiner from one hand to the other, some beyond the thread and others between it and the box, taking care to have those which fall beyond the thread a trifle larger than those which fall inside of it. The operator's hands should be entirely out of the patient's sight. If binocular vision exists, the patient will almost always give a correct answer as to which side of the thread the object falls: if not, nearly half the answers will be wrong.

FIG. 24.—Bar reading.

Javal's bar reading is a fair test for single binocular vision (although there are those who claim that it is really more a test of rapid alternate binocular vision.) The simplest means of using the test is a flat metal bent spring, 1/2 inch wide, to be held by the thumb on the page of the book which is being read (Fig. 24). The idea is to discover whether the patient can read continuously without bobbing his

FIG. 25.—Remy's Diploscope.

head. If there is amblyopia in one eye the patient cannot of course read that part of the print which lies behind the arm of the spring without moving his head: if both eyes have good visual acuity but do not work together, there must either be a head movement or a hesitation when the deviated eye has to suddenly take up fixation, followed immediately by another

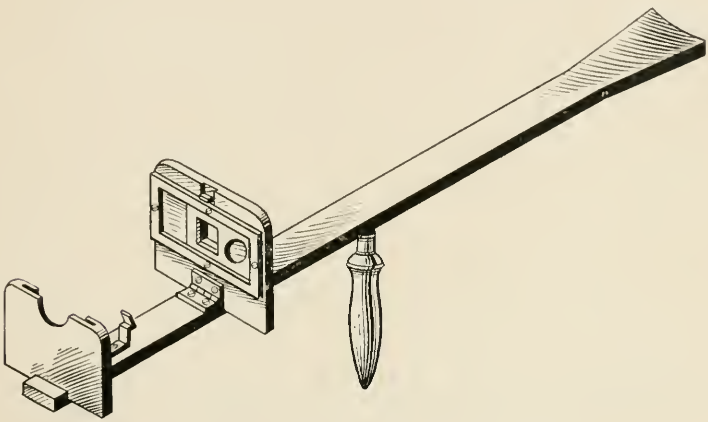


FIG. 26.—Bishop Harman's diaphragm test.

pause when the first eye again takes up fixation. Remy's diploscope (Fig. 25) and Bishop Harman's diaphragm test (Fig. 26) are both evolutions of this same test. Both are of value in the examination of the presence of binocular single vision and both will be referred to in the chapter on the treatment of heterophoria or strabismus.

PART II.

STRUCTURAL ANOMALIES; PALSIES.

OCULAR PALSIES.

Ocular palsies are of intracranial or of orbital origin. The intracerebral areas, invaded by various lesions, include the highest cortical centers, the centers for associated movements, the fibers connecting these centers with the oculo-muscular nuclei in the 4th ventricle, the nuclei themselves, and the trunks of the nerves to the ocular muscles anywhere from the 4th ventricle to their point of escape from the cranial cavity into the orbit. Lesions of the nerve-trunks consist in degeneration of the nerve itself, or in interruption of their function by disease in their neighborhood, such as meningeal exudate, thickening of the periosteum, neoplasms, hemorrhages and wounds; also by vascular changes, such as atheroma, aneurysm, emboli, and thrombi. The underlying cause is generally to be found in constitutional disease, pre-eminently syphilis and tuberculosis. Strikingly causative in this class are tabes and general paralysis of the insane, the rheumatic diathesis, basal meningitis, and brain tumors. Other conditions productive of ocular palsies are the various forms of intoxication, diabetes, nephritis, diphtheria, influenza, hysteria and many of the rarer diseases of the central nervous system. The most recent addition to the causes of ocular palsies is the injection of various anesthetic solutions into the intraspinal canal (so-called spinal anesthesia for surgical purposes). These palsies appear about two weeks after the intraspinal injection is made and behave very much like postdiphtheritic palsies.

Orbital palsies arise from some local cause usually situated at the entrance of the nerve into the orbit through the superior orbital fissure, and are sometimes due to an affection of the nerve itself, at other times the result of pressure upon or disease adjacent to the nerve. There is a class of palsies not caused by

influences acting in the orbit but on the nerve within the muscle, which, by Mauthner and some others, are termed "peripheral," and are ascribed to rheumatism, although Mauthner himself says that such palsies are not infrequently the forerunners of some serious central nervous disease, and are always to be regarded with suspicion. It is our own belief that if many of these cases were followed out for a number of years until their real origin became apparent, we would hear less of "rheumatic" palsies. Congenital ocular palsies may be:

1. Traumatic, from forceps delivery.
2. Inflammatory, affecting the nerve directly or indirectly.
3. Neoplastic, due to cranial or orbital tumors.
4. Teratologic, due to arrested development.

Ptosis.—An exhaustive study of paralysis of the upper lid would be out of place in a monograph on affections of the motility of the eye, yet a brief statement of the principal features of ptosis would seem to be appropriate. On page 7 it was stated that the superior rectus and the levator palpebræ are connected by bands of connective tissue just anterior to the meridian of the globe. Therefore paralytic affection of one will be manifest by partial or complete loss of function of the other.

Ptosis is congenital or acquired. Congenital ptosis may be: of any intensity, but is generally incomplete and double. The lightest cases are those in which, through hypertrophy, abnormal length or loss of contractility, the elevation of the lid is restricted. In the severer grades the smooth lid hangs over the ball, is without folds and can be elevated only by the aid of the frontalis. Congenital ptosis is usually associated with a deficiency in upward movements of the globe, both through congenital defect in the elevators and non-use. Since the lid covers the eyeball no need arises for turning the cornea upward. It may also be complicated by other muscular paralysis, (for example, all of the 3d nerve muscles), by diminution in visual acuity and by nystagmus.

In Wilbrand and Saenger, is given (1st Abt., page 83) a series of instances of the hereditary form, collected from the literature. These authors state that it is almost invariably associated with other

defective muscular action, but does not involve the iris or the ciliary muscle. The affection is passed down through both sexes, and may skip a generation. It is more common among the Jews than among other peoples. They found the causes to be underdevelopment of the levator, bifurcation, union with other muscles, abnormal insertion, and absence or lack of development of the nerves, or the nuclei of the related cortical regions (temporal fissure and angular gyrus).

Acquired Ptosis.—During birth, instrumental delivery may be responsible for producing edema or hemorrhage within the skull

FIG. 27.—Ptosis after meningitis. Patient of Dr. James H. McKee.

FIG. 28.—Bilateral ptosis with complete ophthalmoplegia externa chronica.

by which the nerves in their origin or course are damaged; or by pressure upon the muscle within the orbit; from lesion of the levator induced by wounds to the muscle or its tendon, by pressure, by tumors, by intracranial diseases and in many affections of the central nervous system, notably posterior sclerosis and multiple sclerosis. The lesion is to be sought in the cortex of the brain, in the nuclei, and last of all in the nerves (Fig. 27). It also accompanies or follows infectious diseases and other states such as diphtheria, toxemia, and influenza. It is usually double except in orbital lesions, gradual in onset, of prolonged course, and incurable without operation (Fig. 28).

Ptosis is the most frequent variety of congenital palsy. Paralysis of the external rectus is second in frequency. Marina mentions as quite frequent, conjugate convergence paralysis, the in tegrity of the internal recti being preserved. Mauthner and Dufour consider external ophthalmoplegia (paralysis of all the extrinsic muscles) as a nuclear disease; Möbius regards it as a special infantile disappearance of the nucleus, and recently Simmerling has found true disappearance of the nucleus with degeneration of some of its fibers; at times it is complicated with palsy of the facial nerve. Inability to turn the eye upward has often been found to co-exist with congenital ptosis and autopsies have shown absence of the superior rectus in many such cases. Perhaps similar defects lie at the bottom of many other congenital paralyses.

SYMPTOMATOLOGY.

The main symptoms by which ocular palsies reveal themselves are: 1. Limitation of movement; 2. false fixation; 3. diplopia; 4. vertigo; 5. vicarious rotation of the head.

Limitation of movement in the direction of action of the affected muscle is shown when a test object held in the median line is moved toward the field of action of the palsied muscle. In abducens palsy for instance the sound eye follows the movement easily as far as the limits of the field of binocular fixation, while the cornea of the squinting eye will be seen to stop at the median line; or if it can be rotated further in that direction, it will be by a series of jerks and irregular contractions.

In a case of partial paralysis, the limitation of movement may be so slight as to be almost invisible. The globe, no longer under the control of six muscles, is rotated from the primary position to a secondary one, the resultant of the combined action of the five sound muscles. This deviation may be objectively determined roughly by inspection, and accurately by the use of the perimeter or Stevens's tropometer, and subjectively by the degree of prism necessary to displace the image of the affected eye from its false position back into the vertical or horizontal line, or to fuse it with the true image. Except in frank 6th nerve

or 3d nerve palsies, very little information can be gained by the observation of the limitation of movements, because all the eye movements are under the control of more than one muscle. Therefore, paralysis of one muscle may be, in part, compensated for by the action of its synergist. Again, the arc of the circle of upward and downward rotation is normally much less than the lateral, and imperfect movement is less easy to detect in vertical than in horizontal palsies.

Primary and Secondary Deviation.—The cornea is rotated by the sound muscles in the direction opposite to the affected muscle, the degree of rotation and forced immobility depending upon the completeness of the paralysis and the amount of secondary contraction of the antagonist muscles. This is known as "primary" deviation. If now the sound eye is covered, it will be seen *under the cover* to have assumed the squint corresponding to that of the affected eye. This "secondary" deviation is more pronounced than the primary, since the extremely strong innervation necessary to stimulate the paralyzed muscle involves overaction of these muscles in the sound eye acting with it in associated movements. For the reasons given above, the rotation of the cornea cannot be observed with accuracy in the case of paralysis of the superior and inferior recti and the two obliques, and oft-times we cannot derive much advantage from this measure.

False Fixation or Orientation.—Objects in space cannot be accurately located with the squinting eye because their images do not fall upon the fovea, but upon a neighboring portion of the retina, in a direction and at a distance from the fovea depending upon the muscle paralyzed and the degree of the paralysis. The test instituted by v. Graefe, and called by him the "touch test," consists in closing the sound eye and at once requiring the patient to point toward a designated object in the field governed by the muscle involved. Instead of indicating the true position, the finger will point to some other part of the field governed by the affected muscle. When the sound eye is closed and the patient attempts to walk to a designated object, he tends to a course lying in the field of the paralyzed muscle. In cases of long stand-

ing, these tests are worthless, because the patient has learned to use his judgment and to make allowance for false projection.

Diplopia.[1]—This is the most constant, persistent and annoying symptom of all paralyses. Two images (the true and the false) of every object are seen in that part of the field into which the affected eye cannot be turned. The discrimination by the patient of the true from the false image is not always possible, and other senses than that of sight must be called upon to aid in the discrimination. The physician is aided in his diagnosis by: 1, the relative position of the two images; 2, their relative movability when the test-object is carried in all directions; 3, the loss of parallelism of the vertical or horizontal axes of the two images, this loss becoming more marked in the periphery of the binocular field; 4, the conscious fixation, or the patient's testimony as to which is the true and which the false image. In some cases all tests fail.

Vertigo depends upon false projection and diplopia. It is most annoying in the early stages of the paralysis and with few exceptions becomes less and less troublesome as the case wears on. It disappears promptly and entirely upon excluding the deviating eye from the act of vision, and partially upon excluding the sound eye. The mental disturbances are at times so great that the patient is discouraged from his usual pursuits, and shuns society, seeking seclusion and darkness.

Vicarious Rotation of the Head.—In order to supply the function of the silent ocular muscle, the head is rotated by the action of compensating neck-muscles. By this means the false image may be made to fuse with the true one over a much larger portion of the common field than if the head is held in the primary position, and considerable diplopia and mental confusion are avoided. The secondary head position depends upon the muscle involved. In paralysis of an elevator, the eye is turned down and the head therefore thrown backward on its horizontal axis; if a depressor, the reverse; if the right externus, the head is turned to the right on its vertical axis; if the right internus, to the left;

[1] It is assumed that monocular diplopia is not present.

if the left externus, to the left; if the left internus, to the right. While these compensatory rotations do not specifically determine the muscle affected, they are valuable adjuncts to the diagnosis, particularly if the elevators or depressors are involved.

DIAGNOSIS.

In the diagnosis of ocular palsies the art of diagnosis by exclusion is carried to an utmost nicety. With this purpose in view it is well to bear always in mind certain factors, namely: 1. The law of projection. 2. The doctrine of corresponding retinal points. 3. The synergistic action of the muscles. 4. The antagonistic action of the muscles. If these are well learned and remembered there will be little need for the numerous mnemonics or memory helps offered by many authors. All these four factors have been previously gone into, but the student will do well to refresh his mind about them before attempting to diagnose any ocular palsy. Another series of facts of value in approaching this subject is the grouping of the ocular muscles according to their predominant action. For instance, of the twelve ocular muscles it will readily be seen that four of them are lateral rotators—namely, the two external and the two internal recti; also that there are four whose action is predominantly to elevate the globes—namely, the two superior recti and the two inferior obliques; and that finally there are four which principally depress the globes—namely, the two inferior recti and the two superior obliques. They may be arranged as follows:

LATERAL ROTATORS.

Right and left external rectus.
Right and left internal rectus.

ELEVATORS.

Right and left superior rectus.
Right and left inferior oblique.

4

DEPRESSORS.

Right and left inferior rectus.
Right and left superior oblique.

The convenience of this arrangement is apparent at a glance when one reflects that if a patient complains of diplopia on looking to the right or left, one of the four muscles in the Lateral group must be at fault. If on the other hand the complaint is of diplopia on looking downward, some one of the four Depressors is under suspicion; while if the complaint is of diploia on looking upward the Elevator group will furnish the affected muscle.

These groups are again divided into pairs, one muscle of each pair being in the right eye, the other in the left eye, as for instance the right external rectus and the left internal rectus—or the right superior rectus and the left inferior oblique. (See the chapter on Associated Muscles).[1]

So that with the muscles divided into three groups and six pairs, one should be able by a simple differentiation (the character of the diplopia) to find just what group, then what pair, and finally what muscle is involved in a case of palsy.

If, for instance, a patient comes complaining of double vision on looking to the right, the group of lateral rotators must be at fault, and as the diplopia is greatest in the right field either one of the two right rotators (dextraverters) is under suspicion. This narrows the diagnosis down to the right external or the left internal rectus and the final decision as to which of these two is affected turns on whether the diplopia is crossed (heteronymous) or uncrossed (homonymous). In the former case it is the internus, in the latter the externus that is palsied.

In palsy of the vertically acting muscles, much stress has been laid in former years on whether the accompanying vertical

[1] The term associated antagonists (borrowed from the German) is an unfortunate one in that it is likely to create in the mind of the student the idea that the muscles are not only associated, but also antagonistic. The latter is not true. They are associated, but not antagonistic—hence our belief that the use of the term should be discontinued.

diplopia was also crossed or uncrossed; but in recent years it has been plainly shown that extraneous factors may be at work that may easily confound the practitioner if he relies on this phenomenon too much. Study of the vertical character of the diplopia, alone, will lead to a certain diagnosis. Given, therefore, a case in which the patient complains of double vision on looking down, the depressor group must be interrogated. We know that either one of the inferior recti or superior obliques is underacting. The next step is to find which eye beholds the lower image, for if a depressor muscle is palsied or paretic, the eye to which it belongs cannot be rotated downward as far as its fellow, and therefore its axis pointing higher will see the false image lower than the true one. For illustration let us assume that the image belonging to the right eye is the lower one. We then know that the palsied muscle is in this eye and that it must be either the right inferior rectus or the right superior oblique. The final decision as between these two turns on whether the area of greatest vertical diplopia is down and to the right or down and to the left. If the area of greatest vertical diplopia is down and to the right, it means that the inferior rectus is involved and that the antagonistic muscles have rotated the right eyeball up and to the left and that therefore by the law of projection the area of greatest diplopia must be down and to the right. If the area of greatest vertical diplopia in this same given case were down and to the left (instead of down and to the right) it would imply that the eyeball had been rotated up and to the right by the antagonistic muscles (as in palsy of the superior oblique) and that consequently the area of greatest vertical diplopia must be down and to the left.

The student is recommended to draw a diagram as in Fig. 29 and indicate the amount of lateral or vertical diplopia present in the various portions of the binocular visual field. If a note is made in the margin as to which is right and left and whether the images are crossed or uncrossed and in the case of vertical palsies which image is the higher, the diagram can be studied after the patient has left the surgeon's consulting room and a certain diagnosis arrived at. Thus the accompanying diagram would

indicate palsy of the right externus (Fig. 29). If the images were crossed instead of uncrossed as noted to one side, the diagram would indicate a palsy of the left internus (the associated muscle) (Fig. 30). A splendid means for determining not only the relation of one image to the other but of demonstrating obliquity of one or the other is to place before each eye a compound Maddox

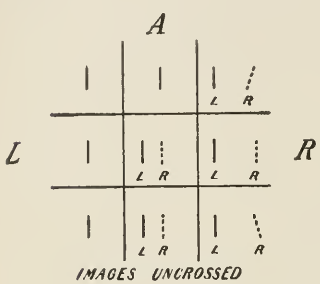


Fig. 29.

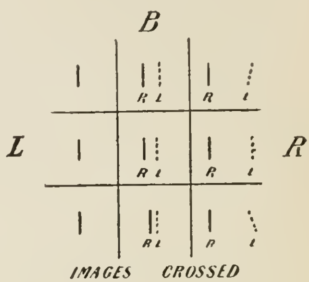


Fig. 30.

rod, one of red color, the other without color. In this way, the amount of torsion present is easily read off from the trial frame and the progress of the case toward recovery may be accurately followed and registered.

SPECIAL PARALYSES.

Paralysis of the Oculomotor Nerve. 1. *Complete Ptosis.*—The abnormally smooth upper lid lies over the ball unmoved by the levator. It can be partly raised by forcible contraction of the occipito-frontalis and the superior rectus. That the partial elevation is due in great part to the former muscle can be readily demonstrated by pressure upon the eyebrows, which will prohibit action of the frontalis. The patient will endeavor to compensate for the lost action of the levator by throwing his head backward.)

2. *Deviation and Limitation of Mobility.*—All movement of the ball, excepting that dependent upon the superior oblique (4th nerve) and the external rectus (6th nerve) is abolished, and the cornea is rotated outward and slightly downward. The physi-

ologic action of the superior oblique alone is well shown in this paralysis. The effort made by the patient to turn the eye farther out and down results in slight actual movement down and out, and the upper end of the vertical meridian of the cornea will be plainly seen to turn toward the median line.

3. *Dilatation of the Pupil.*—The dilatation is moderate and is due to the paralysis of the circular sphincter pupillary fibers. Further dilatation can be obtained by the instillation of a mydriatic, which empties the blood-vessels of their contents, and possibly stimulates the sympathetic fibers and relaxes the choroid generally. The pupil is absolutely unresponsive to light admitted directly or consensually through communicating fibers from the 3d nerve of the opposite side, also to the stimuli of convergence and accommodation (the response to the associated action cannot, of course, be elicited in paralysis of the accommodation and internus).

4. *Failure of Accommodation.*—In emmetropia the inaction of the ciliary muscle (cycloplegia) consequent upon complete 3d nerve palsy does not diminish the previous acuity of vision for distance; in hypermetropia the amount of loss depends upon the degree of defect; in myopia of less than 1 diopter it has no appreciable effect. In estimating the near point, in E. and H. and in low degrees of M. the tests should always be made while the far correction is worn, or its kind and degree known and allowed for. The estimation of the loss is readily determined by restoring the reading power with a glass of known focal length and then measuring the distance from the patient's eye that the print can be read. Thus in E. $1+3$ will allow reading at 13″; in H. of 2, $+5$ would be necessary; in M. of 3 D. the test type would be held at 13″ (without -3); in M. of 5, -2 would restore the reading power for 13″.

5. *Diplopia.*—The false image is on the side opposite to the affected muscle, *i.e.*, crossed and on a level with the true, excepting in the extreme periphery, when it may be slightly higher and tilted away from the true one. Wishart[1] states that when the image of the affected eye is straight and that of the sound eye tilted, nuclear lesion of the same side as the palsied eye may be

[1] Journ. Nervous and Mental Diseases, Dec., 1897.

diagnosed, because as the fibers of the 3d nerve diverge to the nucleus, a few of them pass through the raphe and cross over to the nucleolus for the inferior oblique in the opposite nucleus, and the inferior oblique of the affected eye thus escapes involvement, while the inferior oblique of the sound eye is palsied. On the other hand, tilting of the image of the affected eye indicates a lesion of the *trunk* of the nerve, and the inferior oblique of the affected eye suffers along with the rest of the 3rd nerve muscles of that eye. The images separate from each other the farther the test-object is carried toward the limit of the fie'd governed by the paralyzed muscle. In paresis, the diplopia begins as soon as the middle line is crossed, but in total paralysis the field of diplopia passes beyond the median line, and, in old cases, attended with secondary contraction, single vision is unattainable. The slight elevation of the false image is caused by the contraction of the superior oblique of the affected eye. In the middle portions of the field the vertical turning of this muscle is concealed by the stronger action of the externus in turning the cornea outward; but when the abducting power of the latter is exhausted, the vertical action of the superior oblique becomes manifest.

RECURRENT OCULO-MOTOR PALSY.

1. Ophthalmoplegic migraine. Preceding or accompanying the recurring motor paralyses are attacks of amblyopia, scintillating scotoma, hallucination and pain. Its characteristic symptoms are, as implied by the name: 1, pain; 2, paralysis. The pain begins suddenly in the region of the head supplied by the first two branches of the 5th pair of nerves of one side, principally the first, is intense, of variable duration and usually terminates in reflex vomiting. The paralysis, usually of the 3d nerve, is complete, involving all its branches, and continuing with decreasing severity for several weeks or months. Recovery is usually complete, especially in those cases in which the intervals between the attacks are short and the attacks themselves are brief. The paralysis may become permanent and extend to other ocular muscles.

2. Recurrent oculo-motor palsy appears and disappears at

irregular intervals from childhood to adult life, the paralysis generally increasing in severity and duration with each attack, until frequently, when the patient has reached manhood or womanhood, the palsy is complete and permanent. It is always unilateral and always affects the same nerve. It may be an early symptom of locomotor ataxia or one of the symptoms of central syphilis and brain tumor or a local disturbance in the vascular supply of the 3d nerve nucleus in the anterior part of the floor of the 4th ventricle. It is not amenable to any known treatment

Recurring and partial ophthalmoplegia interna is characterized by partial or complete paralysis of the iris and ciliary muscle. For a period of days or weeks the pupil is dilated and unresponsive to light and the stimulus of associated action, and the accommodation is in abeyance. The symptoms entirely disappear, to recur at irregular intervals. The disease is probably nuclear in origin, and may be an early symptom of central nervous disease, such as locomotor ataxia or multiple sclerosis. Treatment other than local applications of eserin seems to have no influence.[1]

PARALYSIS OF INDIVIDUAL BRANCHES OF THE MOTOR OCULI.

Internal Rectus.—Inward movement of the cornea past the middle line is limited or abolished. The face is turned toward the opposite side, the diplopia is crossed and is found in the field governed by the paralyzed internus, in which direction the false image separates more and more from the true one. The two images are on a horizontal line over the major part of the diplopic field. At the extreme periphery the false is slightly higher than the true image, and its vertical meridian is tilted away from it. For example, paralysis of internus of the right eye: the cornea is turned outward and cannot be rotated inward past the median line; rotation in all other directions unrestricted; the false image is to the left of the true, on a level with it, and separates from it the farther the test-object (candle-light) is carried to the left, where it

[1] This disease was first described by Hansell in the Ophthalmic Record for April, 1898.

becomes slightly tilted, its upper extremity outward. In order to compensate for the abnormal divergence of the eye, the face is turned to the left on its vertical axis; diplopia commences in the middle line; or, in old cases, where secondary contraction of the externus has set in, diplopia is seen over the entire field. Paralysis of the internal rectus, unassociated with palsy of some or all of the others supplied by the 3d nerve of this same side, or with the opposite internus (paralysis of convergence) is extremely rare. The lesion may be a change in the muscular structure itself, congenital, idiopathic, traumatic, or due to an exceedingly fine nuclear disease.

Superior Rectus.—When the gaze is directed upward, the sound eye follows the test-object, but the cornea of the affected eye stops at the horizontal line and the efforts to continue its upward turning result in divergence. Diplopia is found in the upper half of the field of vision. The false image moves farther from the true as the light is carried upward, the former indicating the affected eye by its more rapid movement and greater elevation. Its upper end is tilted toward the temporal side. It is above and crossed. The vertical distance is greater in abduction, the obliquity in adduction, and the horizontal separation diminishes to each side. The head is tilted backward on its horizontal axis, and slightly toward the paralyzed side. In diagnosing paralytic from functional disease of the superior rectus, the extent of the field of diplopia is an important feature. In paralysis, the false image leaves the true below or at the horizontal line. Again, in paralysis, efforts to turn the eye upward result in a rotation upward and outward. The field of greatest diplopia or vertical separation is up and to the right for the right eye, and up and to the left for the left eye. In functional disease, the limitation of rotation is not manifest, and can be determined only by careful measurement with the perimeter or tropometer. The onset of paralysis is sudden; it is preceded or accompanied by headache of a few hours or days and may be associated with symptoms of some general disease. A history of traumatism or disease of the nervous system may be elicited. In functional disease complaint

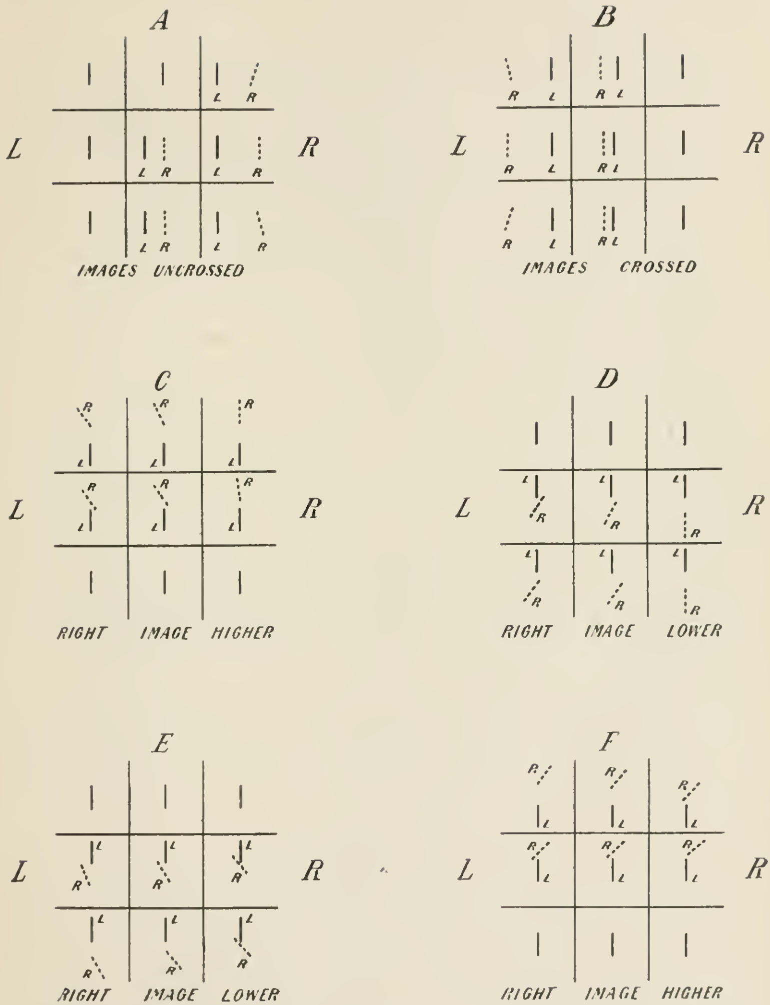


PLATE I.—Position of the images in palsy of the individual muscles of the right eye. *A*, Right external rectus; *B*, right internal rectus; *C*, right superior rectus; *D*, right inferior rectus; *E*, right superior oblique; *F*, right inferior oblique.

is seldom made of diplopia, and the patient will probably give the history of long-continued headache, asthenopia, and reflex nervous symptoms.

Inferior Rectus.—The movement of the eye downward is limited, especially in abduction. The eye is lightly diverged, and the diplopia is in the lower half of the field, the lower image belonging to the affected eye, with its upper end tilted toward the temple. As the eye is adducted, the distance between the images decreases. The head is tilted upward and toward the affected side. In abduction, the false image becomes more tilted, the tilting diminishing when the object is moved to the side opposite the affected eye. The field of greatest diplopia is down and to the right for the right eye, and down and to the left for the left eye.

Inferior Oblique.—The elevation of the eye is limited, especially in adduction, although no deviation of the eye is apparent. Diplopia is in the upper half of the field, the false image, tilted temporalward, is above the true and on the side of the diseased eye. The vertical difference between the images increases and the inclination of the false image decreases upon looking upward and toward the sound side. The field of greatest diplopia is up and to the left for the right eye, and up and to the right for the left eye (Plates 1 and 2).

For the prognosis and treatment of paralysis of the oculo-motor nerve, and of the individual muscles under its control, see the last two paragraphs in this chapter.

TROCHLEARIS PALSY.

A patient presenting paralysis of the right superior oblique muscle will complain of diplopia on looking down. It will be found that the lower image which belongs to the affected (right, for example) eye, is inclined to the left, and that the vertical distance between the images increases as the eye is depressed and adducted. Abduction will increase the obliquity of the image. The head is tilted toward the healthy eye and the face is turned down and toward the affected side. The limitation of movement of the eye will become apparent when extreme down and inward

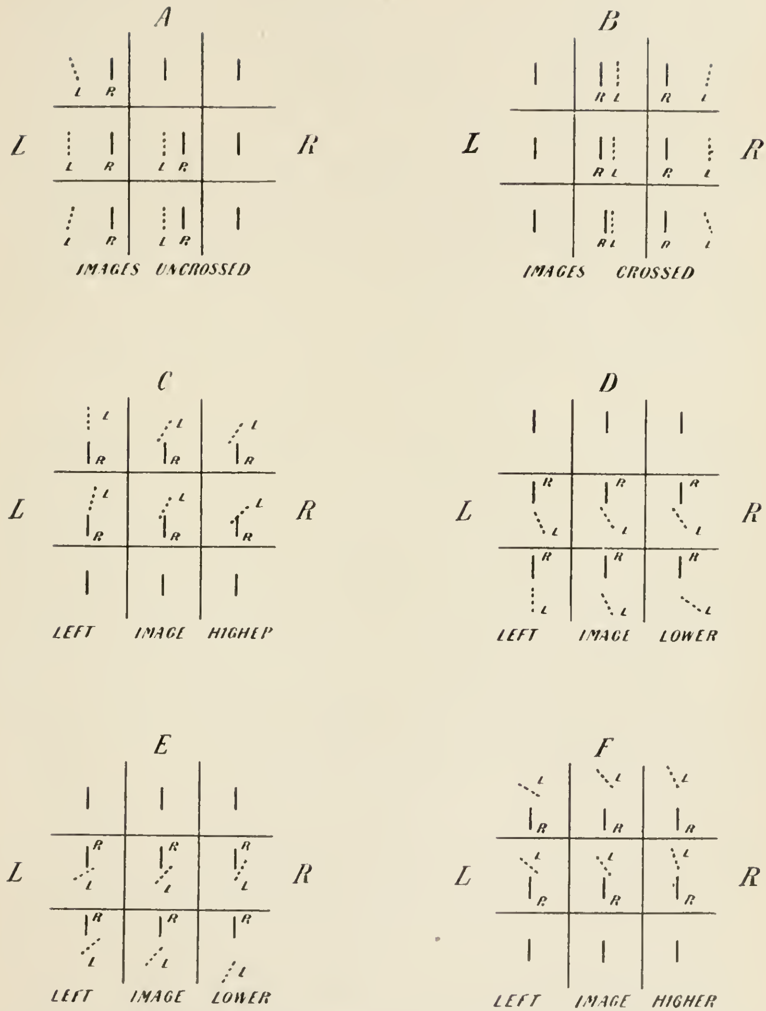


PLATE 2.—Position of the images in palsy of the individual muscles of the left eye. *A*, Left external rectus; *B*, left internal rectus; *C*, left superior rectus; *D*, left inferior rectus; *E*, left superior oblique; *F*, left inferior oblique.

rotation is attempted, and on looking down the patient will see things lower than they ought to be. The field of greatest diplopia will be down and to the left for the right eye, and down and to the right for the left eye. If palsy of the superior oblique muscle occurs as an isolated paralysis, the patient can, by inclining the head downward and to the right or left, produce fusion of the images; and if the palsy be a transient one, it may thus never come to the notice of the surgeon. However, isolated trochlearis palsy as a focal symptom is rather uncommon. The trochlearis is frequently palsied, in conjunction with muscles of the 3d nerve group.

The features of individual paralysis of the superior oblique are well shown in the following history taken from our records.

March 6, 1896. J. M., aged forty-three. For three months he has had diplopia—one image above the other. V. R. 20/50, partly, L. 20/200; low myopia and astigmatism; full vision with correction; no disease of the media or eyegrounds. Covering the right eye with the blue and the left with the red glass and testing with the candle flame, the red light separated from the blue in the lower field, the upper end tilted toward the blue, always homonymous in the left field, the distance between them increasing the farther the candle was carried down toward the right. When the image had reached 20 degrees to the right, the false image became crossed over, and when carried to the left it crossed at 30 degrees. The movement of the eye down and in was limited. The diplopia when first noticed was vertical. A prism of 6 degrees base down, combined with 1 degree base in, in front of the left eye, fused the images in the median field. The diagnosis in this case rested between paralysis of the inferior rectus and paralysis of the superior oblique. That is, since downward movement of the left eye was limited, evidently one or both of the depressors must have been affected. The normal action of the superior oblique is to turn the cornea down and out, with the upper end of its vertical meridian tilted inward; of the inferior rectus, to turn the cornea down and in, with the upper end of its vertical meridian out. In paralysis, therefore, of the superior oblique, the eye

would be turned in rather than out, and the images would be homonymous, as in the above case. In paralysis of the inferior rectus, movement down and in would be limited and the images would be crossed. In both cases the false image would be tilted toward the true. With the field of greatest diplopia down and to the right and the left image the lower, the left superior oblique was plainly shown to be the affected muscle. The lesion, in this case, may be basal or nuclear. It is impossible to tell exactly its character, since the paralysis remained an isolated one, without complications. The patient had a syphilitic history, but did not improve under large doses of iodid of potassium and bichlorid of mercury, nor did the addition of strychnin improve the symptoms.

ABDUCENS PALSY.

1. The constant sign is *homonymous lateral diplopia*, for objects at all distances. The distance between the images widens as the object is carried away and to the side of the affected muscle in the horizontal plane. In the oblique positions, for example, up and out, when the upper ends of the vertical merdian incline toward each other or diverge from each other. This tilting is slight and unimportant for the diagnosis, since it may also be due to complicating paralyses or unequal insertions of the muscles. The false image is on the side of the affected muscle (homonymous). It moves, while the true image remains fixed, the increasing separation of the two images being always due to the movement of the false.

2. *Limitation of Movement.*—In complete palsy the cornea cannot be rotated outward past the median line.

3. *The cornea* of the affected eye is inclined toward the median line—convergent squint.

4. *False Projection.*—Images are not formed on the fovea, but on the nasal side of the fovea—hence the image is projected into the temporal field and the brain is misled as to its position.

5. *Secondary deviation* inward, of higher degree than the primary.

6. *Vicarious Rotation.*—The face is inclined toward the right,

in palsy of the right externus; to the left, in palsy of the left externus.

The long course of the 6th nerve at the base of the brain renders it peculiarly liable to arrest of function during disease, or from traumatism to the skull. Indeed, the external rectus leads in the order of frequency of individual ocular palsies, the order being external rectus first, then unilateral oculo-motor palsy, paralysis of the superior oblique, the inferior rectus, the superior rectus, the internal rectus, and the inferior oblique. Duane (Knapp's *Arch. of Ophthal.*, 1894, p. 61) would award to the superior rectus second instead of third place. Convergent strabismus will be simulated as the eyes are moved toward the palsied side.

Abducens palsy is commonly seen after fracture, or accompanying a syphilitic or other inflammatory process at the base. It also occurs with diseases of the pons. Peripheral or orbital abducens palsy, when not rheumatic in character, is said by Wood to indicate syphilis in adults and tubercular disease in children. However, in the latter connection, it must not be forgotten that abducens palsy in children is not infrequently congenital, and may also be traceable to forceps delivery (Marina).

If paralysis of the external rectus is not peripheral, basal, or pontine, the lesion resides in the nucleus, the cortex, or the connecting tracts, in any of which cases, if the origin be not specific or traumatic, the prognosis is highly unfavorable and ominous of evil for the near future.

The treatment of this particular palsy differs in no wise from that of the other ocular paralyses, outlined in the chapter on Treatment.

The following instance of isolated paralysis of the external rectus is taken from our records:

Case 1.—January 30, 1896. J. S., aged sixty-five, widower, engineer of a shifting or yard engine. Has always been an unusually healthy man, no subjective or objective testimony of rheumatism, syphilis, or tabes. Four days ago while at work on his engine, objects began to appear double, and the confusion became so great within an hour that he was compelled to quit

work. The anterior ocular segment of both eyes was normal; pupils 4 mm., and regularly round; V equals O.D. and O.S. 5/36. With 1.00 D.S. equals 5/6, ocular movements were smoothly and completely executed in all meridians save in movement to the left, when the left rectus externus refused to carry that eye more than 5 mm. beyond the median line.

The ophthatmoscope showed clear media and normal fundi. With a red glass before the right eye, there was shown homonymous double vision, the distance between the images increasing as the candle was carried to the left. There was no upward or downward displacement of the false image, nor was it tilted. A 30 degree prism, base out, before the left eye fused the images at 4 meters; but the patient, even by the fourth day, had learned that by turning his face toward the left he could fuse images and avoid diplopia. Patient was ordered 1/16 grain of biniodid of mercury, and strychnin in increasing doses three times a day. Urinalysis, made by his general medical adviser, shows neither sugar nor albumin, but reveals an excess of uric acid.

Four days later, fuses images with 11 degree prism; twenty-eight days later, fuses images with 5 degree prism.

The patient did not return for further observation. The fact that the patient had been a railroad man all his life, that the palsy was in all likelihood non-focal, and that it disappeared so rapidly under biniodid of mercury, would indicate that the foregoing was an instance of specific abducens palsy.

Case 2.—Abducens paralysis due to traumatism of the skull. M. J., aged twenty-five, applied at the Polyclinic Hospital, December 15, 1897, complaining of constant diplopia. He stated that on December 8, while skating, he fell and struck the left side of his head on the ice. He was dazed for a short time, but recovered and noticed nothing unusual until evening, when objects became double and remained so. The right eye was converged; the cornea could not be moved past the median line; there was homonymous diplopia on the right side of the field. Media and eye-grounds normal. The lesion was probably basal and consisted of minute hemorrhage either in the sheath or pressing upon the trunk of

the 6th nerve on the right side. No cerebral symptoms developed and there were no other complications. This, then, is an instance of traumatic paralysis occurring in the long course of the 6th nerve, at the base of the brain, from hemorrhage due to traumatism of the left side of the skull. The final outcome of the case is unknown.

Inasmuch as the nucleus of the 6th nerve is said to be the center for conjugate deviation, a few words may here be said of

CONJUGATE PALSIES.

Inability to move the eyes in association to the right, or, as the case may be, to the left beyond the median line, is sometimes seen in individuals who nevertheless converge close up to the ordinary convergence near point. This paralysis of synchronous action of one internal rectus and the external rectus of the other eye is known as conjugate palsy, and may follow upon lesions in the cortex, the corona radiata, the internal capsule, or the pons. It is not an uncommon symptom in gross lesions of the cerebrum and the pons, although the conjugate palsies secondary to cerebral lesions are exactly the reverse of those seen in diseases of the pons. Swanzy (Diseases of the Eye, fourth edition) contrasts the association of the palsies with their casual lesions as follows:

Cerebral Lesions.	*Destructive.*—Eye turned away from palsied side.
	Irritative.—Eyes turned toward convulsed side.
Pontine Lesions.	*Destructive.*—Eyes turned toward palsied side.
	Irritative.—Eyes turned from convulsed side.

Wernicke (Nervous Diseases, by Dercum) speaks of conjugate palsy of upward and downward movements of the eyes as being secondary to disease of the corpus striatum and of the optic thalamus.

PARALYSIS OF CONVERGENCE.

A woman, aged twenty-four, was under the care of Dr. Dercum, at the Jefferson Hospital, on account of inability to walk. Examination revealed that the eyes and their appendages were

normal in every respect, with the exception that the patient had lost absolutely all power of convergence. She could follow with both eyes a test-object moved to the right, left, above, or below, the optic axes remaining parallel throughout all these movements; but when the test-object was moved in the median line from the distant to the near point, she altogether failed to follow the movements with either eye. She had no diplopia in any direction, and no ocular paralysis. The optic axes remained parallel in all movements, hence the image of the test-object fell on corresponding parts of each retina. The symptom probably arose from cerebellar disease.

CONJUGATE VERTICAL PARALYSIS.

March, 1895, J. B., aged thirty-five, complained of diplopia. Vision, right 20/30; left hypertropia 6 degrees, exotropia 4 degrees, from paralysis of the right superior oblique. There was, in addition, *complete paralysis of upward movement of both eyes.* An object held above the horizontal line could not be followed by either cornea, and efforts to look up resulted only in partial contraction of the levator palpebræ. In April, 1897, divergence and upward deviation of the left eye marked; vision of the right had fallen to 20/40, but remained full in the left; ophthalmoscope showed commencing atrophy of the right optic nerve; in the left retina, the arteries were smaller than normal, but not markedly so; both fields concentrically contracted for white and colors, right decidedly; pupils responsive to light and accommodation, but sluggish. In October, 1897, vision in both eyes had fallen to 20/50; left eye diverged and rotated slightly downward; upward movement abolished as before, downward movement limited; optic nerve atrophy had further advanced. There was also incoördination of extremities, and walking was on this account difficult. The knee jerks were exaggerated. This is a case of advancing ophthalmoplegia and optic nerve atrophy, the symptoms of which were preceded, for some years, by loss of upward movement in both eyes. The patient had no history of specific infection or constitutional disease.

PROGNOSIS.

Prognosis in ocular palsies hinges on whether the paralysis be: 1. Acute or chronic; 2. individual or associated; 3. traumatic or idiopathic;[1] 4. due to a central or peripheral lesion.

It is further influenced by the nature of the general disease-process of which it may be a symptom.

An acute individual palsy, if traumatic or peripheral, offers in many cases a reasonably hopeful outlook. Especially is this the case if the patient is the subject of a traumatism, or of syphilis, rheumatism, or tabes. In the latter case, the palsy is likely of a peripheral nature, and though the prognosis as to the paralysis itself is favorable, it is important to remember that a transient external ocular palsy may be an early and significant symptom of locomotor ataxia, and should never be disregarded. Acute palsies of higher origin (basal, nuclear, or fascicular) are to be regarded with apprehension; and though they often prove transitory, they more frequently portend intracranial mischief—syphilitic, hemorrhagic, inflammatory, or neoplastic in character—and thus become symptoms of great value in estimating the probable nature of obscure central lesions.

An acute associated paralysis, or conjugate palsy, while of itself of no special importance, and though not infrequently transient, is like the foregoing, ominous for the future, and should invariably lead to thorough-going investigation of the functions of the patient's nervous system. It is occasionally hysteric in origin.

Chronic paralyses, of whatever character, are gloomy affairs, promising little for the present and less for the future. If the palsy be an individual or isolated one, it is apt to be accompanied by secondary contraction of the antagonistic muscle of the same eye. Some people live through a long and busy career with palsy of one or more of the extrinsic eye-muscles, and suffer little or no discomfort therefrom after the acute stage is passed, but this is exceptional. Oftener some grave nervous malady unfolds its features, and the case drags its weary length along, or in the midst

[1] Idiopathic, here used in the sense of a paralysis occurring without demonstrable lesion in a previously healthy person.

of life the patient may be in death. Such sudden termination is the rule in paralyses occurring in arterio-sclerotic individuals.

TREATMENT.

As to treatment, ocular palsies arrange themselves in two classes: those in which the origin is more or less apparent, offering opportunity for rational treatment, and those in which the obscurity surrounding the case compels empirical therapy.

Fortunately for both surgeon and patient, this phase of the subject is much simplified by the aid of statistics, which show the predominant influence of syphilis in the production of paralyses of the ocular muscles. According to Alexander (Syphilis und Auge), 59.4 per cent. of all ocular palsies are specific, the oculomotor being most frequently selected by the disease. Vigorous anti-syphilitic medication is, therefore, indicated in all ocular palsies that are not traumatic, or cannot be shown to be distinctly rheumatic, tubercular, or gouty; and the caprice of the surgeon will largely dictate what shall be the mercurial or iodin preparation used, likewise the mode of its application. Moreover, mercury or iodin will be preferred as the palsy is a secondary or tertiary syphilitic phenomenon; and it is well to bear in mind that all medication aimed at ocular palsies (and specific treatment in particular) is more likely to be fruitful of results if assisted with Turkish baths and hot packs, and other such measures as will promote increased functional activity of the skin. By this means most heroic internal treatment will often be generously tolerated. Recently salvarsan has been extolled as of high value in syphilitic palsies but as 2 or 3 cases of ocular palsy are alleged to have followed upon its use it will be wise to wait for complete and satisfactory demonstration of its unfailing usefulness. It is well to bear in mind that the very first thing to be done in the treatment of any ocular palsy is to have the patient wear a blinder over the eye to which belongs the palsied muscle or muscles. This will immediately relieve the nausea and headache due to the diplopia and false projection.

Isolated palsies occurring in adult life in an apparently healthy

middle-aged individual should invariably arouse suspicion of, and thorough search for, tabes, or general paresis which, if the suspicion be confirmed, calls for anti-syphilitic treatment in fully 90 per cent. of all cases (according to Erb and Fournier). Every such suspect deserves careful interrogation of the pupil reflex to light and accommodation, and of the patellar tendon reflexes.

Essential rheumatic palsies (a class that is yearly growing proportionately smaller) yield generally to salicylates or iodids, the former in the acute, the latter in the chronic forms of the disorder. Other rheumatic remedies have been favorably mentioned from time to time.

When paralysis of an ocular muscle is the result of encroachment of a tubercular focus upon the nucleus or trunk of the nerve supplying the palsied muscles, treatment is of little avail; happily, such conditions are rare.

Palsies of gouty origin are commonly the peripheral manifestations of an obliterating endarteritis or hemorrhage in that part of the floor of the 4th ventricle in which the 3d, 4th and 6th nerve nuclei are located. If the arterio-sclerosis be not too advanced, much benefit and even complete recovery may follow upon the active use of absorbents and diaphoretics, at the same time guarding well the heart with digitalis or strychnin. This treatment is also applicable to the palsies associated with the emboli and thrombi common to cardio-vascular disease.

The treatment of palsies secondary to fracture of the cranium, either in the fronto-motor region of the calvarium or at the basis cranii, is embraced in the general treatment of the fracture itself—namely, rest in bed, antiphlogistics and absorbents, or surgical interference. The same management holds good for the palsies seen with orbital or sphenoidal fractures. If the nerve trunk or trunks be lacerated, treatment will accomplish little, while antiphlogistics and absorbents will go far toward restoring function if its arrest be due to the pressure of hemorrhage or inflammatory exudate. For those palsies that accompany inflammations or tumors of the brain and its membranes, the treatment must be aimed at the major affection. If the process be a specific

one, its therapy is simple. If it prove tubercular, rheumatic, gouty, arterio-sclerotic, senile, or neoplastic, symptomatic treatment only is called for, and will often prove unavailing. Palsies resulting from the introduction of the various local anesthetics into the spinal canal (spinal anesthesia for surgical purposes) have a spontaneous tendency to recover. The externi are most frequently involved. Three to six weeks usually ensue before binocular single vision is restored.

There remains to be mentioned in the therapy of these conditions, strychnin and electricity.

The value of strychnin in restoring palsied ocular muscles has, perhaps, been over-estimated. This may be due, in part, to the fact that it has been exhibited indiscriminately in all forms of palsy, whether cortical, fascicular, nuclear, or peripheral. It is in the latter class only that it is of marked value, although its use is admissible in the acute stages of almost any palsy. A method that is highly esteemed is to order 1 gr. of strychnia in 1 ounce of water, beginning with a 10-drop dose (gr. 1/48), three times daily, and increasing the dose 1 drop each day until toxic symptoms appear.

As to electricity, faradism is most popular. A strength of 1 mp. should never be exceeded. If, as some prefer, the slowly interrupted constant current be used, 3 mps. is all that will usually be borne with comfort; the cathode to be used over the closed lid and the anode (or indifferent pole) over the corresponding temple or mastoid. The quantity of current that really passes through the affected muscle is uncertain. Some surgeons prefer to place the anode on the sclera directly over the palsied muscle after cocainizing the conjunctiva.

It is ofttimes necessary, in addition to the medical treatment of the paralysis itself, to relieve the annoyance which the diplopia and vertigo bring with them. If the palsy be slight, amounting only to a paresis, prisms which fuse the images—and are properly placed in an ordinary spectacle frame—may be worn, and the patient thus rendered comfortable until such time as the cure may be accomplished. When the diplopia cannot be thus over-

come, it is, as we have already said, best to exclude the paralytic eye from all efforts at binocular vision by placing before it, in a spectacle frame, a piece of ground glass.

In practising the mechanical treatment suggested by Michel, the eye is cocainized and the conjunctiva seized near the insertion of the palsied muscle, when the eyeball is drawn forcibly as far as possible beyond the limit of contraction and then back again. Michel recommends daily use of the movements for about a minute each time.

In a general way it may be said that most ocular palsies are from two to twelve weeks in recovering, so that the surgeon will do well to be guarded in discussing this phase of the question with the patient.

In long-standing paralyses, operation offers the best results. Section of the antagonist, combined with advancement of the affected muscle and adjoining capsule and conjunctiva, affords the latter the most favorable mechanical conditions for work. This presupposes, however, some slight degree of contractility, consequently complete paralyses are incurable by operation.

SPASM OF THE OCULAR MUSCLES.

Primary spasm of individual ocular muscles has been described from time to time by various writers. This must be distinguished from the over-action of a muscle that results from over-development in the muscle itself or from its insertion too near the cornea so that its mechanical purchase is abnormally increased. Primary spasm is rare indeed. It has been recorded as occurring in chorea, meningitis and other irritative brain lesions. The spastic oculomuscular conditions occurring in hysteria never affect single muscles.

Secondary spasm giving rise to the phenomenon in ocular palsies known as secondary deviation is common enough and at times quite marked.

NYSTAGMUS.

Nystagmus is a disturbance of associated movements tremor-like in character. It may be lateral, vertical, rotational (rotary

or wheel-like or torsional) or irregular. The binocular variety is usually met with although about sixty cases of the unilateral variety have been recorded. The rapidity of the excursions varies from 5 to 300 or more per minute. They are best studied through the ordinary ophthalmometer, but any of the numerous corneal microscopes answer quite as well. The principal symptom is poor vision, with sometimes vertigo and compensatory movements of the head. The main causes are defects in the transparency of the media—congenital defects (spasmus nutans), occupational (miner's nystagmus) ataxic conditions—labarynthian disease and certain central nervous diseases, such as Friederich's ataxia and preeminently multiple or disseminate sclerosis. The treatment will be determined by the etiologic factors—operation when feasible when the defects in the media are remediable, changes of occupation, treatment of labarynthian disease, etc.

PART III.

FUNCTIONAL ANOMALIES.

HETEROPHORIA.

HETEROTROPIA.

HETEROPHORIA.

GENERAL CONSIDERATIONS.

In the study of the relation of the muscles to each other in states of abnormal tension and relaxation (but without paralysis), we leave the clearly defined field of recognized symptoms and conditions, and enter that of uncertainty, vagueness, and speculation; we pass from the logical results of anatomic changes to the confused manifestations of unknown psychologic disturbances. We do not deal with demonstrable lesions of the muscular or nervous system, but with *tendencies* to incoördination, the result of innervational peculiarities that thus far have been untraceable by the dissector's knife or physiologist's microscope. We are brought face to face with the ocular complications of disordered health, of inherited or acquired abnormal susceptibility, of illogical and uncommon results of common causes, of individual idiosyncrasies—in a word, of unbalanced nerve-action. While it is not disputed that functional muscular anomalies follow known causes according to known principles of cause and effect, it is maintained that some of the most difficult problems offered for solution cannot be explained by any satisfactory theory. Obstacles surround the examination and treatment of every case and can only be overcome by repeated trials with modern diagnostic methods and the persistent applications of physiologic and conservative remedies. The *individual* element must not be lost sight of, and every factor, ocular and extraocular, that can have a bearing on the solution thoroughly investigated. The oculist is not the refractor or the tenotomist alone, but is the physician as well.

Functional deviations are so intimately bound up with the refractive status as to compel frequent mention of the latter in discussing the various forms of muscular imbalance. Indeed,

no problem in heterophoria is free from the complicating influence of the action of the ciliary muscle, whether the eye be emmetropic or not. The importance of this relation is so great that some authorities maintain today that all muscular "imbalance" proceeds from errors of refraction, and that the persistent wear of correcting lenses will ultimately dissipate the heterophoria. Functional deviations of the eye depend, further, for their solution upon heredity, age, sex, social condition, temperament, physique, vocation, hours of work, mental worry, and numerous other minor factors. It will thus be readily seen that they are not so simple in their causes and treatment as has been suggested by some authors. Many of the older writers looked upon such deviations of the ocular muscles as symptoms of a lowering of the general nervous tone (whims of the nervous system, as some have called them), or of some general pathologic process; and although later methods and up-to-date instruments of precision have plainly shown that a goodly number of heterophorias are not symptomatic, but are substantive conditions attended by a train of their own symptoms, the view of these older writers was not without foundation, and it deserves particular emphasis in this day of exceeding specialism.

Muscular Balance.—The eyes may be said to be swung and held taut in the orbit by a delicate muscular harness and the orbital fascia. The four recti muscles in combination tend to draw the globe backward into the orbit, which action is opposed in greater or less degree by the tendency of the superior and inferior oblique to bring the globe forward. Hence, a slight degree of contraction of all the muscles is necessary to maintain the globe in the proper position during all waking hours. This process, simultaneously active in both orbits, produces coördination of the visual axes, so that when the eyes are directed toward an object, their axes will meet exactly in that object, and the eyes are said to be balanced, or in equilibrium.

It is important to remember in this connection that the direction of the visual axes when the eyes are in the *anatomical* position of rest (resulting from the form of the orbit, the insertion of the

optic nerve and the natural length of the muscles when not innervated), is generally divergent, rarely parallel, and hardly ever convergent. But the function of normal eyes never allows independent deviation, hence the parallel direction demanded for distance can never be abandoned while we are awake, and the unconscious innervational habit pulls the eyes from the *anatomical* position of rest to parallelism, which is the *functional* position of rest; *functional*, because the function of the eyes has produced it, and *position of rest*, because the position assumed by habit and unconscious innervation is free from all exertion.

Any disturbance of the factors just mentioned will cause muscular imbalance, and yet want of muscular balance is not incompatible with perfect binocular vision, for, in many cases, visual axes that tend to deviate are brought back to parallelism by increased innervation to a given muscle or group of muscles, and it is this necessary extra outlay of nervous energy that frequently brings on asthenopia in small degrees of muscular imbalance.

Functional disorders of coördination are manifest or latent. There may be an *actual turning* of one optic axis from the other, involving a loss of associated movement, or the want of coördination may show itself as nothing more than a *tendency* to deviation. Between these two extremes of want of equilibrium (the one representing the accomplished strabismus, the other latent overaction or underaction of synergist muscles) is a middle stage, characterized by the development of either the deviation tendency or the squint, according to the demand made on the different muscles; for example, the insufficiency of the internal recti for near work, described by v. Graefe forty years ago. The study of this subject will be simplified if the student will bear in mind that functional want of equilibrium concerns in all cases the destruction of the normal relation between two sets of opposing muscles rather than the insufficient or overaction of any one muscle. Thus, in esophoria we are dealing with the power of convergence, and not the strength of the internal recti. It is unscientific to attempt to separate the muscular from the innervational apparatus, the more so as the latter comprises both the fusion-force and

the impulse conveyed by the nerve-trunks; and equally futile is it to speak of one muscle as having more power, as measured by prisms, than its corresponding muscle in the other eye.

The various gradations from the slightest tendency of the optic axes to turn from equilibrium to the highest degree of permanent squint, are but steps in the same direction, or degrees of the one affection, and, in great part, the causes which underlie the one grade are found to produce all the others. For instance, a tendency to turn the visual axes toward each other has, as its fundamental cause, hyperopia. The degree of inward turning or tendency is determined largely by the personal equation. Many hyperopes have absolutely no want of equilibrium, and yet others, even with low grades of hyperopia, develop marked muscular disorders. It has been contended that because hyperopia does not produce a squint in every instance, the squint must have some other cause, but it is well known that the same cause produces different effects according to individual characteristics. A low grade of hyperopia in a nervous or overwrought disposition will give rise to serious reflex neuroses, whereas the same degree of hyperopia in a constitution robust, strong, and resisting, will have no symptoms whatever. Again, a high grade of hyperopia is, in some, perfectly consistent with equilibrium of the muscles, while, in others, it is the cause of decided strabismus. This individuality can only be described in vague terms. It is known as a neurotic disposition, an enfeebled, unresisting nature and susceptibility to disease or an exaggeration of reflex excitability. Therefore, when all circumstances are favorable, reflex neurosis may arise from muscular anomaly, and there is no doubt that, in a few instances, the claims made by enthusiastically credulous writers are justified, but such cases are exceptional in even the largest experience, and the theory that a considerable proportion of the insane, of the epileptic, of the choreic, owe their disease wholly to the existence of either a refractive error or a consequent muscular anomaly, is dangerous and unsound.

In examining patients who have no organic disease of the eye or its environment, but suffer from reflex headaches and annoyances

of various kinds, estimation of the condition of refraction and of the ocular muscles should be among the earliest findings. The investigation of the muscular status should be made both before and after the mydriatics are instilled, because of the direct influence these drugs have in many cases upon both the kind and degree of the defect.

The nomenclature of anomalies of the ocular muscles has received a decided impulse in the past 25 years. For functional deviations Stevens has suggested a classification that has met with quite general acceptance in America and England. It is as follows:

Orthophoria, perfect binocular balance.

Heterophoria, imperfect binocular balance. Heterophoria presents many varieties, namely:

Hyperphoria, a tendency of the visual axis of one eye to deviate above that of the other.

Hypophoria, a tendency of the visual axis of one eye to deviate below that of the other.

Exophoria, a tendency of the visual axes outward.

Esophoria, a tendency of the visual axes inward.

Hyperexophoria, a tendency of the visual axis of one eye to deviate upward and outward.

Hypoexophoria, a tendency of the visual axis of one eye to deviate downward and outward.

Hyperesophoria, a tendency of the visual axis of one eye to deviate upward and inward.

Hypoesophoria, a tendency of the visual axis of one eye to deviate downward and inward.

It must be remembered that, in the functional anomalies under discussion, both eyes are involved and that while the above nomenclature may describe the symptom, it does not locate the lesion. It is simply a clinical convenience. For instance, right hyperphoria means either that the right elevators are too strong for the right depressors, or that the left depressors predominate over the left elevators. In other words, it simply

signifies that one eye tends to turn upward or the other downward, without indicating which is the faulty eye.

To this classification Savage would add cyclophoria, or insufficiency of the oblique muscles; and Duane, hypokinesis, deficiency of action of an individual muscle; and hyperkinesis, excessive action of an individual muscle; and parakinesis, irregular action of an individual muscle.

CONCERNING PRISMS.

All problems that deal with the ocular muscles turn more or less upon the use of prisms. It will therefore be most profitable for the student to consider at this juncture the nature and properties of prisms, so that he may the better appreciate their application to the diagnosis and treatment of functional muscular anomalies.

A prism differs from a piece of ordinary plain glass only in the fact that its two sides are inclined toward each other, forming an edge or angle. Their surfaces are plane (that is to say without any curvature, as is met with in lenses). By reason of this

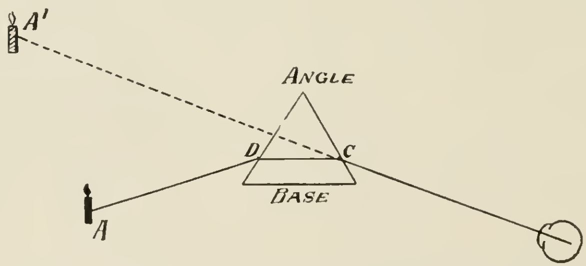


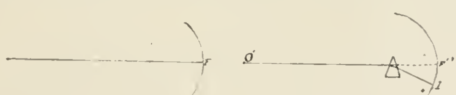
FIG. 31.—Action of a prism on a beam of light. Dotted line indicates direction which the projected beam takes.

inclination of the sides of the prism toward each other a beam of light instead of passing through it without change of general direction (as in the case of window glass) is bent from its course toward the broad end or base of the prism. For the better understanding of the refractive properties of prisms, the student will do well to bear in mind the law which provides that as a beam

of light passes from a rarer to a denser medium it is bent toward the perpendicular, and that as it passes from a denser to a rarer medium it is bent away from the perpendicular. Thus, as in Fig. 31, if the eye be placed at B in the path of the beam after it emerges from the prism, it will not see the candle at A, its true position, but in the direction of the line BC, projected through the prism along the dotted line to A^1. Hence there is displacement of the object in the direction of the angle of the prism and on this phenomenon is based the axiom that *prisms displace the image of an object in the direction of their edge, angle or apex.*

If, when the eyes are directed toward an object 20 feet or more distant, a 10° prism be placed, base in, in front of the right eye, it so displaces the object that the image falls to the inner line of the fovea, and, unless the eye by rotation of its cornea temporalward effects a corresponding inward displacement of the fovea centralis, diplopia is inevitable. The above elementary principle may be illustrated in its application to the prism test for determining heterophoria or orthophoria.

In Fig. 32, O is the object, OF the ray proceeding from the object to the fovea, F, of the right eye (R), and O^1F^1 the ray from



Right retina (R). Left retina (L).

FIG. 32.

the same object which if not interrupted by the prism would fall upon F^1 in the left eye, but a prism placed base *down* before that eye refracts the ray to a portion of the retina beneath the fovea. Since the lower half of the retina refers its impressions to the upper part of the field, O^1 will be projected above O to O^2 and in a vertical line with it in orthophoria, or to one or the other side of O in lateral heterophoria. To test for hyperphoria or hypophoria the refracting prism is placed with its base in, producing insuperable lateral diplopia with image of O refracted to the nasal side of the fovea of that eye before which the prism is placed;

if hyperphoria exists, one image will be seen higher or lower than the other.

According to the law of projection, if an image falls to the right of the fovea the object O will be projected or seen to the left portion of the field; if to the left, to the right portion of the field; if below, to the upper; and if above, to the lower portion of the field. In Plate 3, *A* is the fundus of an eye before which no prism is placed; *B*, fundus before which prism is placed base *down*, *I* representing the new position of the image in the lower half of the retina; *C*, prism base *up; I* displaced to the upper half of the retina; *D*, prism base *in*, displacing *I* to nasal half of the retina; and *E*, prism base *out*, displacing *I* to temporal half of the retina.

The Relation Between Accommodation and Convergence.

If there is one set of phenomena that more than any other will help the beginner in ophthalmology toward the solution of some of the obscure problems in refraction and imbalance of the ocular muscles, it is the understanding of the relation between accommodation and convergence.

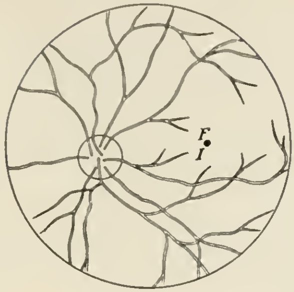
CONCERNING ACCOMMODATION.

Since the time of Donders, it has been known that if an individual who has been shown (by the use of a mydriatic or cycloplegic) to be emmetropic, fixes the gaze on an object at infinity, there is no accommodation called into play. If the same individual be requested to look at proper sized print at a distance of one meter, it is equally well known that one diopter of accommodation is called into play.[1]

Similarly, to focus proper sized print at a distance of half a meter (50 centimeters or 20 inches) two diopters of accommodation

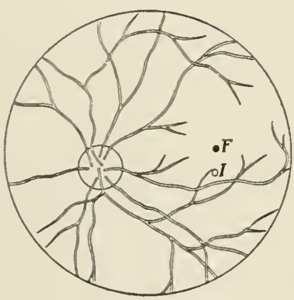
[1] This is proven by again using a cycloplegic in the eye of such a patient, when it will be found that he can no longer read at one meter the print he read easily at this distance when the eye was not atropinized. To enable him to read the print at this distance a plus one diopter lens must be placed before the eye, thus proving that this was the amount of accommodation exercised before the cycloplegic was used.

Fig. A



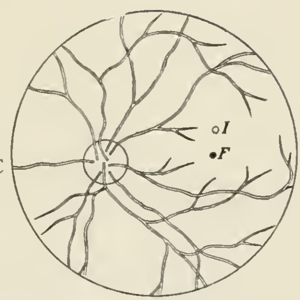
F = fovea. *I* = image. No prism. *I* falls an *F*.

Fig. B



Prism base *down*. *I* falls below *F*.

Fig. C



Prism base *up*. *I* falls above *F*.

Fig. D

Prism base *in*. *I* falls to nasal side of *F*.

Fig. E

Prism base *out*. *I* falls to temporal side of *F*.

PLATE III.

must be used by the patient, and to focus the same size print at a distance one fourth of a meter (25 centimeters or. 10 inches) four diopters of accommodation are demanded. It will be readily seen, therefore, that if a patient who is hypermetropic 2 diopters, focuses on print at 1 meter's distance, he will employ 3 diopters of accommodation (that is to say the 1 diopter the emmetrope

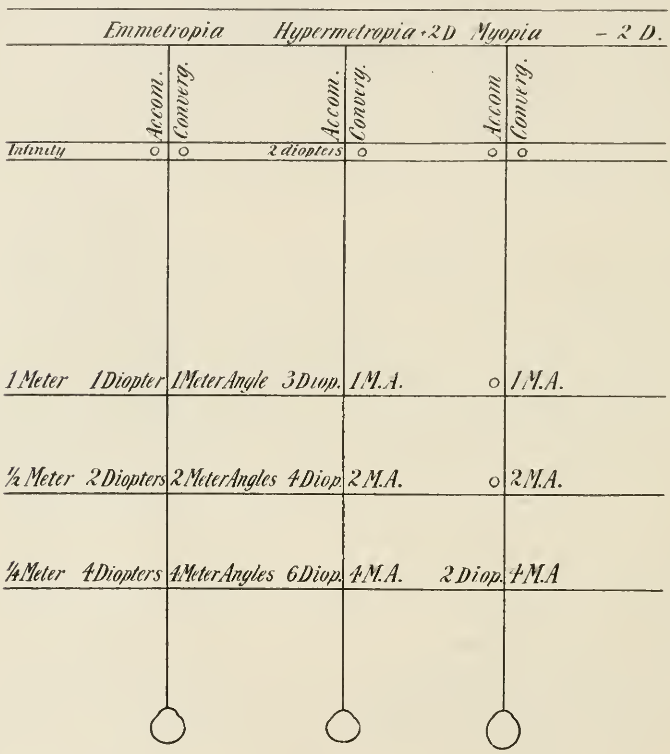


FIG. 33.

would exercise, plus the 2 diopters the hypermetrope exercises for infinity); and at one-half meter's distance, the two diopter hypermetrope would utilize 4 diopters of accommodation, while at one-fourth meter's distance, the 2 diopter hypermetrope would require 6 diopters of accommodation to see the print clearly. In the case of myopia of 2 diopters, there would of course be no

effort at accommodation at infinity, nor at one meter's distance. Neither would there be at one-half meter's distance, for 50 centimeters represent the far point of such an eye and no accommodation is necessary to enable the patient to read easily. At one-fourth of a meter, however, for which point the emmetrope accommodates 4 diopters, the myope of 2 diopters would naturally exercise but 2 diopters of accommodation. The accompanying diagram may help to a clearer understanding of the principles involved (Fig. 33).

Concerning Convergence.

If vision were effected by means of one eye only, there would be no point from beyond infinity up to within a very short distance from the eye to which we could not adjust the dioptric system of that eye. But man generally sees simultaneously with two eyes, and the direction that the eyes must give to their lines of fixation in order that they may be simultaneously directed toward a point of fixation anywhere inside of infinity, is called convergence. For some years after Donder's exposition of a precise method of estimating accommodation at various distances it was realized that when an emmetrope accommodated for an object 13 inches or 35 cm. distant from the eye, he also converged his eyes on that point, for if he did not he would surely see double. But no scheme of accurate and practical measurement of the convergence was in vogue until Nagel (Graefe and Saemisch Handbuch, 1st edition, Vol. VI, Chap. X) offered his ingenious unit angle known as the meter-angle. This is now commonly designated as MA.

Convergence is in this way easily expressed. For instance if a patient bring his gaze from infinity to an object 1 meter distant, he is said to have converged 1 meter angle. If the object be 1/2 meter (50 cm.) distant, he has converged 2 meter angles; if 1/4 meter (25 cm. or 10 inches) distant, he has converged 4 meter angles. The following table may help to an understanding of the principles involved.

Accommodation diopters	Convergence meter angles or MA	Distance at which tests are made
0	0	Infinity.
1	1	1 meter.
2	2	1/2 meter.
4	4	1/4 meter.

It will now be readily observed that in emmetropes there is a very regular and close relation between accommodation and convergence. If such a patient at 1 meter accommodates 1 diopter, he also converges 1 meter angle. If at 1/2 meter he accommodates 2 diopters, he also converges 2 meter angles; if at 1/4 meter, he accommodates 4 diopters, he converges 4 meter angles.

Let us assume, however, that the patient is hypermetropic 2 diopters. A new relation now appears, for if such a patient surveys an object 1 meter distant he *converges* the regular 1 meter angle, but he *accommodates* 3 diopters (the 1 diopter that the emmetrope would employ plus the 2 diopters he must need call into play even for infinity). If he fixes his gaze on an object at 1/2 meter, he converges 2 meter angles and accommodates 4 diopters; and if the object is at 1/4 of a meter he converges 4 meter angles and accommodates 6 diopters as shown in the diagram (Fig. 33).

Let us now assume for further illustration that the patient is myopic 2 diopters. Another relation now develops. If such a patient views an object 1 meter distant, he converges 1 meter angle, but he does not accommodate at all, as 1/2 meter (or 50 cm. or 20 inches) is the far point of accommodation in such an eye. If the object viewed is at 1/2 meter, the patient will converge 2 meter angles, but will still not accommodate at all, as this is the far point of the eye. If the object is at 1/4 of a meter, the patient will converge 4 meter angles and accommodate only 2 diopters, as shown by the diagram (Fig. 33).

The presence of astigmatism in combination with any spherical

error will naturally only complicate this relation; (the student who wishes to go deeply into the physics, mathematics and psychology of this relation is referred to Nagel's original article (*loc. cit.*) and to Landolt's voluminous work on the "Refraction and Accommoadtion of the Eye," translated by Culver). Enough has been said, however, to show that nature establishes for each person some manner of relation between accommodation and convergence if they enjoy binocular single vision. It will be found to be a most elastic relation subject to many changes and more or less adaptable to varying states of refraction in the same individual. It is well for the student to realize this point, that *it is the breaking in upon this relation (peculiar to each individual) that produces much of the discomfort complained of by most patients when they first put on glasses.* If they have been hypermetropic (say 2 diopters) when their glasses are first worn they naturally need to use less accommodation and therefore there is less impulse to convergence, so that a certain amount of confusion is thrown into the completed visual act. In some individuals who do not readily acquire newly established coördinations and coördinate relations, this period of discomfort may extend into weeks and weeks, in which case it may become necessary to cut down the strength of the plus glass ordered and thus break in less on the relation between accommodation and convergence which had existed prior to putting on glasses. In others but a few days suffice for the establishment of new relations and they quickly become used to their glasses. The same is true in myopia and is even truer in astigmatism. So that unless the ophthalmologist is by nature a persuader of people, he will have much difficulty in carrying many of his patients through this period that is so trying to them, unless he bears in mind all the time the intimacy of this relation and its importance in making the final judgment as to what lenses shall be prescribed; it will suffice to say that it is the experience of the authors that full corrections in hypermetropes are not often well borne, this in spite of the fact that on theoretic grounds the full mydriatic correction should be given, thus establishing that relation be-

tween accommodation and convergence which normally obtains in the emmetrope. As has been already said, however, if the intrusion into the patient's previously established relation between accommodation and convergence is made too great and too abrupt (as it generally is by ordering the full corrections) the patient will finally conclude that the glasses are "*too strong*" and refuse point blank, in many instances, to continue any longer with them.

METHODS OF DIAGNOSIS.

The purpose of the various tests for the determination of muscular imbalance is to so affect the retinal image of one eye, by alterations in color, shape, or position, that the fusion-impulse will no longer be exercised, and that the tendency to deviation will become changed from a latent into a manifest one.

These tests may be arranged in three groups as follows:

1. *Those that displace one image.* (Displacement or diplopia tests.)
2. *Those that distort one image.*
3. *Those that neither displace nor distort either image.*

1. Diplopia (or Displacement) Tests.

The classic test first suggested by v. Graefe is the prism test. A prism of 8 degrees, with its base down, or up, is too strong to be overcome by the muscles it antagonizes, and will produce insuperable diplopia. For instance, to test lateral equilibrium, a prism of 8 degrees held base down before the right eye, with its base-apex line exactly vertical, will so refract the rays entering that eye that they will fall upon the lower portion of the retina, and a false image of the light will be projected into the upper part of the visual field. In orthophoria, the true (or lower) and the false (or upper) lights are in a vertical line. In esophoria, the upper image will be to the right of an imaginary line running vertically through the lower or true light. The prism displaces the retinal image of the light to the lower and (if the eye is turned

in) also to the inner half of the right retina; it is therefore projected, or seen, up and to the right. In exophoria, with the prism in the same position before the right eye, the false image will be above and to the left. The distance that the upper light is to the right or left of the imaginary vertical line drawn through the lower one, is the *linear* measure of the degree of the deviation. The prism (with its base *out* for esophoria, *in* for exophoria) necessary to move the upper image until it is directly over the lower one will be the *angular* measurement of the deviation. The principle of this test is exactly the same as that of all others—namely, the destruction of the unconscious fusion-impulse. The individual seeing two images of the same object simultaneously accepts the impression that the two images represent two objects. Fusion having been thus destroyed, the deviation *tendency* becomes an *actual turning*, and the eyes assume that position which is most restful for them and to which they are impelled by the conditions present in the individual case. The prism commonly employed is of 8 to 10 degrees. This strength is sufficient, not only to produce insuperable diplopia, but to separate the false from the true image by an interval great enough to allow slight deviation tendencies to cause a lateral displacement of the images that is instantly perceptible. Two serious errors arise when higher degree prisms are used—namely, the false image is refracted to a point on the retina so far removed from the real fovea that the findings may not accurately represent the anomaly, and the slightest turning of the prism from its axis will vitiate the result by producing artificial heterophoria. In Stevens' phorometer, instead of a single prism of 8 degrees before one eye, the effect is divided between the two eyes by means of two prisms of 4 degrees each which are held in a frame that can be rotated to test either the horizontal or the vertical tensions. (See Fig. 34.) The instrument is convenient, because both vertical and lateral imbalance can be determined by simply revolving the test-prisms into the horizontal or the vertical meridian. A total of 8 degrees is selected, because, at the distance at which these prisms are placed from the patients' eyes, muscles of average diverging

power cannot fuse the double images. With the instrument fixed to test the lateral muscles, two images separated vertically will be seen. In orthophoria these images are directly in a vertical line, for although single vision has been destroyed, no hitherto latent tendency to turn the eyes in or out has become manifest, and the lateral muscles are said to be in equilibrium. In exophoria or esophoria, one light, instead of being exactly above the other, will assume a position a little to the right or left, because the eyes are now free to respond to the influence of the relatively stronger act (convergence in esophoria and divergence

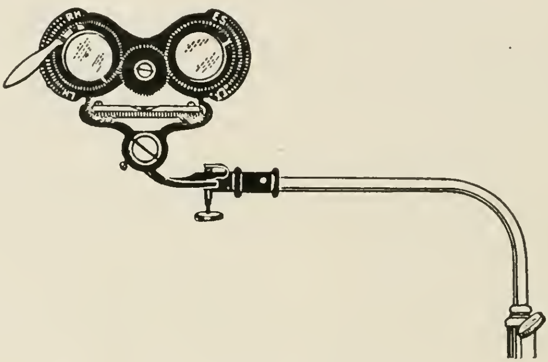


Fig. 34.—Steven's Phorometer.

in exophoria.) The degree of prism now necessary to bring the upper image directly over the lower one, is the measure of the deviation. This is accomplished by simply rotating the lever of the Stevens instrument until the two images are in exact vertical alignment when the muscle's status can be immediately read off from the scale provided for this purpose. The lever is then carried around exactly 90 degrees when the test is quite as quickly made for the state of the vertical muscles (by means of lateral diplopia) and the reading taken at once from the scale. It must be remembered we are dealing, not with one, but with both eyes. For instance, in esophoria we study *not one internus*, but the power of convergence as contrasted with the power of divergence, and by this test we determine, in esophoria, that convergence is abnormally and relatively strong.

The Maddox double prism consists of two 4-degree prisms, bases together, fitted into an ordinary test glass cell and held before one eye (Fig. 35). It will cause the light as seen by that eye to be doubled (monocular diplopia). If now the other eye be uncovered, a third, which is the true image, will appear

FIG. 35.—Maddox double prism.

midway between the two and directly on a line with them in orthophoria (Fig. 36). 1. With the double prism placed in the trial frame two images are seen in the vertical meridian. The true, or middle image, will be out of line, to the right or left, according as there is exophoria or esophoria. 2. Let the two images

FIG. 36.—Effect of a double prism on a beam of light. (Maddox.)

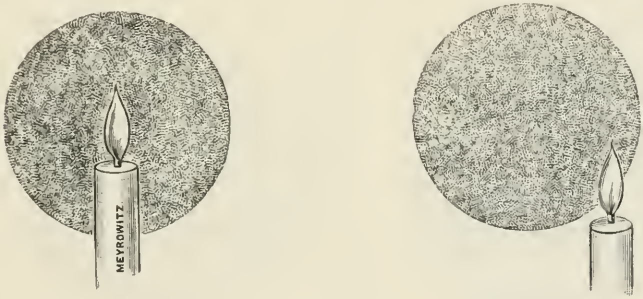
be on a horizontal line, then the true image will be above or below this line in hyperphoria. The test is facilitated when the double prism is ground in red instead of clear glass. Another application can be made of this prism by observing whether the middle or true image is equidistant from the two false (red) ones. If not,

the prism necessary to restore it to the orthophoric middle position will be the measure of both the kind and the degree of the heterophoria. This prism is usually applied to investigating the tensions of the oblique muscles by having the patient survey a line on a card. If they are faulty in action, the middle line is tilted up or down at the right or left.

The Cobalt Test.—A glass stained with cobalt oxide and ground to fit the trial frame has the power of intercepting all the rays of the spectrum excepting the blue and the red. The image seen by the eye before which the cobalt glass is placed will be much smaller than the real image, and will have a red center with a blue halo, or a blue center with a red halo; the former in myopia, the latter in hypermetropia and emmetropia. This test has the advantage over all the others in that it does not refract the rays before they enter the eye, and in all cases the rays pass through parallel and are focused on the fovea. Hence, if deviation be discovered it must be the result of muscular insufficiency and not due to inadequacy of the method of diagnosis. The method is valuable only in cases of 2 degrees or more of vertical and 4 degrees or more of horizontal imbalance. Fusion power is largely destroyed, and in the presence of any considerable heterophoria the patient will see two images, one the clear or natural colored light, the other the smaller blue-red light. In orthophoria both images fall upon the fovea and the composite light will then be the clear light tinged with red and blue. In heterophoria the cobalt image will be separated from the natural image according to the kind and degree of the defect. In esophoria, for example, it will be on the side of the eye covered by the cobalt glass and on a level with the true light, the interval between them depending, as in other instances, upon the degree of the defect. In our opinion, diplopia tests (with the exception of the cobalt test) do not always indicate the true condition, for the reason that the foveal region in each retina is not considered, but rather the fovea in one eye and a point outside the fovea in the other eye. Hence, while the fusion-impulse is destroyed (which is necessary), we are expecting identical muscle-action as the

result of stimulation of different retinal areas in the two eyes. We have found that such tests are likely to show exophoria to be more frequent than esophoria, and have often found a positive contradiction between the results of these tests and those obtained by the use of the Maddox rod and other tests.

The Convex Spherical.—A convex glass of a strength of 15 to 20 diopters, covered in all parts excepting its center, is placed before one eye in the trial frame. The image seen by that eye assumes the shape of a large blur upon the fovea and the adjoining retina. In orthophoria, the clear light seen by the uncovered eye will be situated in the center of the blurred image of the covered eye (Fig. 37). In heterophoria of low grade, the clear



Orthophoria Heterophoria.

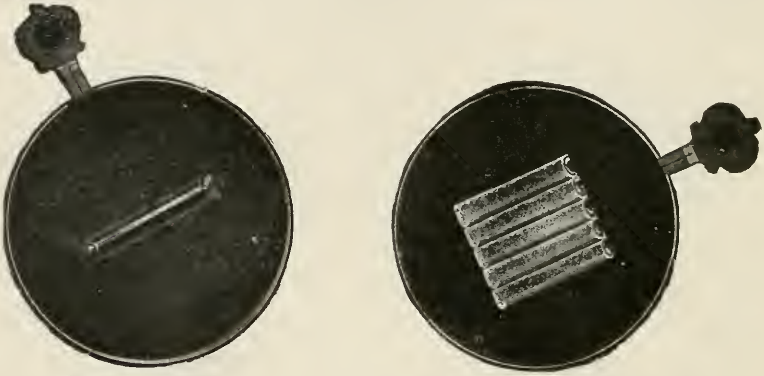
FIG. 37.—The convex spherical test.

image appears on the outskirts of the blurred image, and in the higher grades will be seen entirely outside of it. The space between the true light and the center of the blurred area is the linear measure of the deviation, and the prism required to refract the clear image into the center of the blurred one will be the prism measurement of the heterophoria. This test can be accurate only when but a small portion of the exact center of the lens is left clear, because rays of light passing through it at any considerable distance from the axial ray are refracted away from the fovea, and should an excentral portion of the lens be brought opposite to the pupil, the image of the light will not fall on the

fovea. Hence, the method is open to the objection of the prism test and to the additional one that unless extreme care is experienced apparent deviation may be created by faulty use of the test.

2. Distorting Tests.

The Maddox Rod (Fig. 38).—This consists essentially of a rod of glass conveniently adjusted in a disc to fit the trial frame. When light is refracted by a glass rod, a luminous point becomes a line or streak, since the rod is nothing more than a strong cylinder. As a glass rod refracts rays of light opposite to its axis



Maddox simple rod. Maddox compound rod.

FIG. 38.

the eye will see a streak of light in the reverse meridian to that in which its axis is placed. Hence, if the rod is placed vertically before one eye, the image of the light seen by that eye is a horizontal streak; if placed horizontally the image is a vertical streak. In orthophoria the candle-flame or point of light 6 meters distant to which the patient's gaze is directed is seen as a streak by the eye before which the rod is placed, the streak passing through the unaltered flame seen by the other eye. In heterophoria the streak is seen either to the left, to the right, above or below, according to the nature of the defect. In esophoria the streak is on the same side as the eye which sees

it. For example, a Maddox rod so placed before the right eye that the streak is vertical will be seen to the right of the candle-flame. In exophoria the conditions are reversed, the streak under the same conditions being seen to the left of the flame. In hyperphoria, the phenomenon is a little more confusing. For instance, in right hyperphoria, if the rod is placed before the right eye, the streak will be seen below the flame; if it is placed before the left eye it will be seen above the flame; in left hyperphoria, the rod before the left eye will produce a streak below the flame; before the right eye, a streak above the flame. The prism necessary to change the position of the streak from its faulty position into the light (the orthophoria position)

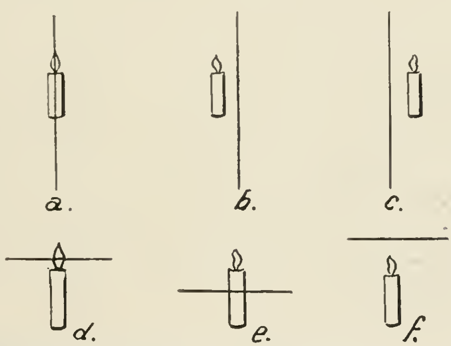


FIG. 39.—Maddox rod before right eye. *a*, lateral balance; *b*. esophoria; *c*, exophoria; *d*, vertical balance; *e*, right hyperphoria; *f*, left hyperphoria.

is the measure of the heterophoria. This instrument is simple, inexpensive, accurate and trustworthy, especially in its latest form of the compound rod, and exceedingly useful *when the student is familiar with the law of projection.* The advantage of the latter over the simple rod is that no special care in its adjustment is necessary; with the simple rod it is essential to accuracy that the rod shall be in line with the visual axis. While the image is greatly distorted by this test, yet the foveal region of the retina is included in the site of the image, and we are studying the relations of the two foveas to each other, and not, as in the prism test, the relation of one fovea with a

circummacular portion of the other retina. By the rod test we find esophoria more common than exophoria, and from the study of other conditions, such as the refraction, are led to believe that this is correct. For the relative positions of the streak and light in the various muscular states, see Fig. 39.

3. **Tests which Neither Displace nor Distort Either Image.**

The Cover Test.—The patient is requested, while standing in a good light, to gaze at a small object 20 feet or more distant. The eyes are screened alternately, thus breaking up fusion. As the cover is carried from one to the other eye, no movement will be seen in orthophoria because the visual axes are parallel, but an excursion will be noticed in heterophoria. As the cover is carried to and fro, if marked esophoria be present, the eye uncovered will move in a direction exactly contrary to the movement of the cover; *e.g.*, if the cover is carried from the left to the right, the left eye will move to the left, and vice versa; and the prism, base *out*, before either eye that stops this movement will be the approximate measure of the heterophoria. The method is applicable to all high-grade heterophorias of whatsoever kind, as well as the prism measure of strabismus. It does not consume more than thirty seconds in its application and should never be omitted from the study of any case.

FIG. 40.—Cover for cover test and parallax test.

The Parallax Test.—The preceding or cover test is entirely objective. If, however, as the cover is carried repeatedly from one to the other eye the patient is asked to state whether the object surveyed seems to jump from side to side or up and down, the test then becomes subjective and is known as the parallax test. The slightest apparent movement of the object will, after a few trials, be remarked by the patient, who accurately describes the

direction and extent of the movement. In orthophoria naturally the object will appear perfectly stationary. If the object moves in the same direction as the cover is carried, exophoria, and if in the opposite direction esophoria, is present. If the object appears to shift downward when the right eye is uncovered there is right hyperphoria, and if the reverse, left hyperphoria. This movement may be completely controlled by prisms, and the degree of prism which stops the movement is the measure of the heterophoria. This test is susceptible of extreme delicacy, deviations as slight as one-eighth of a degree being measurable in some instances. Fig. 40 illustrates a convenient cover with handle for use in this and the preceding test.

In general terms it may be said that those tests which are at the same time the most accurate, the simplest, and require the least apparatus, are the ones most likely to lead the beginner to the surest results. For this reason the Maddox rod (compound) and the cover and parallax test are trustworthy, although the phorometer has many warm advocates. The cover and the parallax test have the great advantage of finding the eyes just as they are and as the patient uses them all day long and is least likely to create any factors of error. On the other hand, the phorometer is easy and quick of application and very helpful to the novice in indicating for him the kind and amount of deviation present.

Tests for the Reading Distance.

The Dot and Line Test.—The principle is exactly that described for the prism test at 20 feet. It was devised by v. Graefe for testing insufficiency of the interni only. A prism of 8 degrees is placed base *down*, before one eye. The patient gazes at a small black dot, through which a line is drawn, on a card held at the ordinary reading distance (Fig. 41). If the prism is placed base *down* before the right eye, the two dots will be seen on the same line in orthophoria. In lateral insufficiency both dot and line will be doubled, the upper belonging to the right eye. The false image will be to the right (esophoria) or to the left

Fig. 41.

(exophoria) according to the lateral deviation. In hyperphoria, with the test-card and the prism changed from the vertical to the horizontal position, the false dot and line will be above or below the true dot and line. In balance of the vertical muscles the false and true dots will be on the same vertical line. In our judgment, the dot alone forms a superior test, since the line maintains, in part at least, the desire for fusion and serves, in a measure, to prevent independent movement of either eye. A small printed word may be similarly employed.

The Maddox test for the reading distance, based also upon prism displacement, provides a scale for the measurement of the angular deviation without other means (Fig. 42).

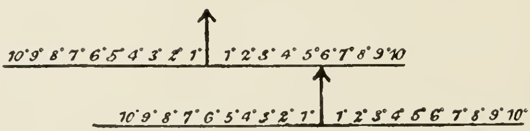


FIG. 42.—Maddox test for the near point.

Maddox Rod and Miniature Light.—In our later experience we have come to rely almost wholly on the Maddox compound rod and the small electric light commonly used in the electric ophthalmoscope. The patient holds the light at his usual reading or occupation distance and the muscle balance at that point is just as quickly, accurately and easily secured as in the test for infinity. Moreover, the moment the test for the reading distance muscle status is completed, the Maddox rod may be removed from the trial frame and the little light used as an ophthalmodynamometer (as described on page 107), which information should always be elicited in cases with obscure symptoms.

MUSCULO-DYNAMICS.

In making up our minds as to the advisability of either optical or surgical treatment of heterophoria the relations of the defective muscles and their synergists to the antagonistic muscles must be given due weight. No tests for muscular coördination are com-

plete that do not take account of the relation to each other of the various conjugate rotations of the globes, the practical determination of which is based upon the ability of the muscles to overcome prisms base up, down, in, or out, while both eyes are fixed on a small object at 6 meters or at the ordinary reading distance. For instance it is not enough to say a patient has 3 degrees of exophoria to induce treatment for exophoria, but this diagnosis should be confirmed by showing a corresponding change from the normal relations of abduction and adduction; that is, a diminished prism convergence and an increased prism divergence. In hyperphoria the diagnosis should be substantiated by an increased supra-duction or corresponding decrease of the infra-duction. Thus right hyperphoria of 2 degrees would indicate that right supra-duction was stronger by that amount than right infra-duction.

If the prism which is placed before the eye is not too strong to overcome the power of fusion (in other words, to destroy binocular vision) the eye before which it is placed will be so rotated that the fovea will be moved from its original position to that occupied by the image. This faculty of setting aside by rotation the transient diplopia thus induced is known as the ability to overcome prisms, or the prism rotation, or the deviation power of the muscles. The student must not confuse prism rotation in the sense just mentioned with the arc rotations of the eye; for instance, the abductors will normally overcome but 8 to 10 degrees of prism base-in at 6 meters (external prism-rotation, abduction, or prism divergence) whereas the eye can be rotated or the gaze directed 45 to 50 degrees from the primary position (arc rotation or version).

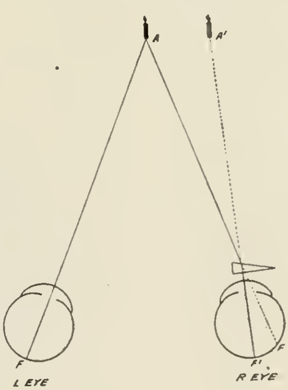


Fig. 43.

When the prism is first interposed the right eye sees the image at A¹ (Fig. 43), but in response to the fusion impulse it is rotated

outward so that the fovea is moved from F to F[1], where the image may fall directly upon it and binocular vision be thus restored.

The limits to the power of rotation of the eyeballs are fairly well defined in the various meridians as measured by the prisms that they overcome. Beyond these limits fusion is no longer possible and diplopia ensues, hence the following terms:

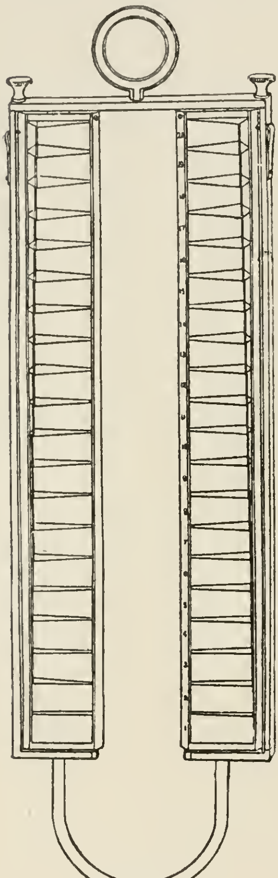


FIG. 45.—Gould's prism battery.

Abduction or *prism-divergence.*

Adduction or *prism-convergence.*

Supraduction or *vertical divergence.*

Infraduction or *vertical divergence.*

To test abduction the prisms are placed base *in* before either or both eyes.

To test adduction the prisms are placed base *out* before either or both eyes.

To test supraduction the prisms are placed base *down.*

To test infraduction the prisms are placed base *up.*

Formerly prisms were made square, but they are now so shaped that they may be fitted into the cell of an ordinary trial frame.

It is the general custom to have the patient seated about 6 meters away from a small electric light or lighted candle, directing him to fix his gaze on the light. Prisms are then interposed with their bases up, down, in, or out respectively until permanent diplopia is produced. The highest prism that can be overcome, or with which single vision is still possible, is the measure of the prism rotation of the eyes —*e.g.*, if the eyes overcome an 8 degree prism, base *in*, but not

a 9 degree, abduction equals 8 degrees; if a 15 degree prism, base *out*, but not a 16 degree, adduction equals 15 degrees; if a 2 degree prism, base *up* or *down* before the right eye, but not a 2 1/2 degree, right supraduction or infraduction respectively of 2 degrees is shown. It should be borne in mind that a prism with its base down before one eye is equivalent in its action to the same degree of prism with its base up before the other eye.

Various devices are employed by different workers for the estimation of the ductional powers of the ocular muscles, principal among which are single prisms, the prism battery, Risley's rotary prism, Jackson's rotary prism, and Landolt's ophthalmodynamometer. The method just described, upon the principle of which most of the others depend, is that by the use of the single prisms. A more convenient method of interposing one prism after another in testing the ocular rotations is by the prism-battery, first suggested by Noyes and later modified and warmly advocated by Gould (Fig. 44).

The series begins with 1-degree prisms on each side, and increases by integers of 1 degree to 20 degrees; thus by steps of 2 degrees each, all degrees of abduction and adduction from 2 degrees

FIG. 45.—Risley's rotary prism.

to 40 degrees may be measured. "Each battery is revolved at pleasure, being fixed by a pivot mechanism above and below, and temporarily held in the position desired, by clutches at the sides. To measure adduction each battery is placed bases *out;* to measure abduction, each battery is revolved so that the bases are in. The open central space permits the lenses to be brought close to the

eyes without interfering with the patient's nose." This instrument is applied only to the lateral rotations. For measuring the vertical rotations, we would suggest a similar battery (single) carrying weak prisms (from 1/4 degree to 5 degree) bases up or down. By reversing the battery both supra- and infraduction could thus be readily and accurately determined. However, all these requirements and necessities are beautifully met in what is known as the "prism mobile," on the principle first suggested by Sir John Herschel, who showed how by placing two prisms in opposition and rotating them in opposite directions, we can produce the effect of a single increasing prism. By far the neatest application of Herschel's idea is that of the Rotary Prism designed by Risley (Fig. 45), to be fitted into an ordinary trial frame. On turning the little mill-edged screw, the two prisms, of 15 degrees each, rotate in opposite directions, the strength of the resultant prism showing on a graduated scale engraved on the

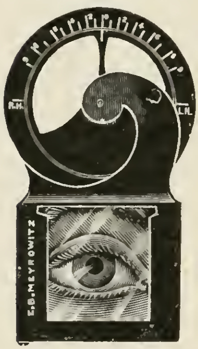


FIG. 46.—Well's hand phorometer adjusted for testing the lateral muscle balance.

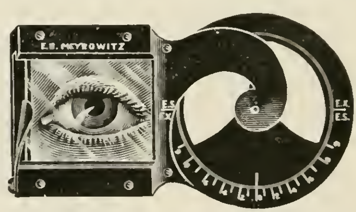


FIG. 46a.—Well's hand phorometer adjusted for testing the vertical muscle balance.

blackened front of the containing cell. However, by occasioning chromatic aberration and prismatic astigmatism, all rotary prisms share the disadvantage of lowering the visual acuity in one eye only, and thus the desire for single vision is more interfered with than if the prism effect were divided between both eyes. Maddox has therefore suggested an apparatus in which one prism revolves before each eye. It is practically the same as the prism arrangement in Stevens' phorometer, and is useful only in measuring lateral rotations. A similar instrument for measuring vertical rotations could easily be devised by having the prisms

fitted into Maddox' device, bases *in*, and then rotating them in opposite directions. Wells offers a very handy pocket phorometer for clinical and bedside use (Fig. 46). To produce more delicate results in the lower prismatic powers, Jackson employs in his rotary prism three prisms, one 15 degree stationary prism and two rotating ones of 7 1/2 degrees each.

MEASURE OF DUCTION POWERS.

Of the four principal prism rotations, above mentioned three of them—namely, abduction, supraduction, and infraduction—are fairly constant in quantity, and authorities agree more or less in fixing the power of:

Abduction	from 6 to 8 degrees.
Supraduction	from 2 to 3 degrees.
Infraduction	from 2 to 3 degrees.[1]

Much depends on the method used in the determination. The above figures are those furnished by single or separate prisms held before the eye one after another. Rotary prisms, especially as employed by Maddox, or any similar apparatus of graduated increment, indicate somewhat higher average powers of rotation, viz.:

Abduction	8 to 10 degrees.
Supraduction	3 to 4 degrees.
Infraduction	3 to 4 degrees.

The fact seems to stand that if, at the first trial of abduction, it is found to equal 7 degrees, there will be little if any variation in its degree, no matter how often it is tried on the same day or succeeding days, provided only that the same method is used each time. The same relative constancy is found in supraduction and infraduction. Not so, however, with adduction, which by its variation from day to day, and even from hour to hour in some cases, has thrown much confusion into the subject of muscular

[1] These figures hold for 20 ft. or more only. For any distance less than 20 ft. abduction would be greater and adduction less; for instance, at 15 ft. abduction equals about 10 to 12 degrees, and if the eyes are steadily approached, a point will finally be reached where abduction and adduction are equal.

anomalies. Perhaps this confusion hinges on the neglect of many observers to distinguish between primary adduction (or the prism convergence manifested at the first trial) and cramped or trained adduction (the prism convergence possible when the patient has learned to temporarily dissociate accommodation and convergence). Moreover, personal equation counts for no little variation in the results of different investigators. Stevens[1] says: "that although an exact standard of adduction is not to be expected, should the adducting ability fail to reach 50 degrees (prism) after a reasonable amount of practice, it is likely to be deficient." Risley [2] foundthe average adduction for 20 feet in 25 non-asthenopic individuals to be 25 degrees; while Bannister,[3] who conducted a series of careful studies on 100 soldiers (all in fine physical condition), found the average adduction for 20 feet to be 14 degrees.

It seems to the authors that Bannister's statistics indicate plainly the average degree of *primary* adduction, while Stevens and Risley's figures have reference more to *trained* adduction. However, it must be borne in mind that convergence and divergence are acts so intimately bound up that it is almost impossible to dissociate them, so that the study of one side of the phenomenon necessarily involves the other. For instance, in overcoming prisms bases in (or fusing lights through prisms held bases in before the eyes), a stimulus to divergence is created. The eye before which the prism is placed is compelled to diverge, in order that a single image of the light may be preserved. It will be noticed that we speak here of divergence and not of the external rectus, because all muscles which help to turn the eye out are really included in the test. When the prism is placed before one eye only, all of the diverging is done by that eye, and the other unwaveringly maintains its direction toward the object focused. Yet it does not follow that the testing is confined to the eye that turns, for the turning eye is held in its divergent position and the

[1] Norris & Oliver, "System of Diseases of the Eye," Vol. II, 1898.

[2] *Univ. Med. Mag.*, January, 1895.

[3] *Annals of Ophthal.*, January, 1898.

other in its straight position by a contraction of all the external muscles, and therefore we are, in a certain sense, testing convergence and divergence at the same time. The completed action is really a very complicated one. For example, a 6 degree prism is held *base in* before the right eye; in order to overcome the momentary diplopia induced by the prism, the abductors of the right eye are called into play and that eye diverged, as can be seen through the prism. Strangely enough, the left eye is held straight, notwithstanding the general rule that movement of one eye in any direction is always accompanied by a similar associated movement of the other eye,[1] and that therefore the adductors of the left eye tend to turn that eye in the same direction as its fellow. To neutralize this latter impulse the abductors of the left eye are called upon and their action necessitates, in turn, action on the part of the adductors of the right eye, which would immediately bring the right eye back to parallelism were not the abductors fully occupied in keeping it in such position as to avoid diplopia. Hence the introduction of the 6 degree prism disturbed the divergence primarily and convergence secondarily. The same phenomenon occurs when prisms are placed bases *out* before one or both eyes. In testing supra- or infraduction a similarly complicated act is excited. Thus: when a 2 degree prism is placed base down before the right eye, the image falls on the right retina below the fovea, in consequence of which the elevators contract and the right eye rolls up, turning its fovea until it reaches the position of the displaced image. Naturally an equal stimulation is sent to the elevators of the left eye (according to the above-mentioned rule for associated eye-movements), but a change in the position of the left fovea would destroy the test so that the upward impulse of the left eye must be met by an equal downward impulse of the right eye, which is prohibitive because of the diplopia that would be sure to follow. Hence, while the elevators and depressors are in equilibrium, they are only artificially and temporarily so. We are in a sense

[1] Movements induced by prisms not too strong to permit fusion constitute the sole apparent exception to this rule.

determining the power of elevation as compared with that of depression, but we have been really investigating the limits of equilibrium by prism stimulation of allied and of antagonistic muscles.

Further knowledge of the relations of convergence and divergence may be gained from the use of Landolt's ophthalmo-dynamometer (Fig. 47). Just as in the study of the accommoda-

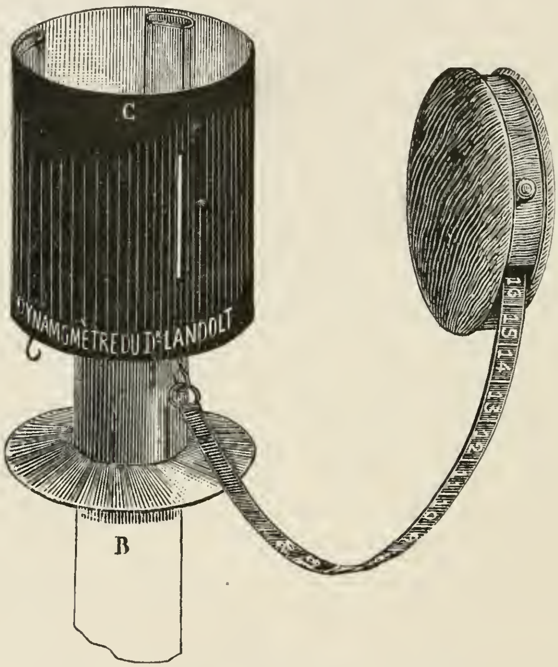


FIG. 47.—Landolt's ophthalmo-dynamometer.

tion, we learn its amplitude by estimating the position of the punctum proximum and the punctum remotum, so in the study of convergence we find that its amplitude is equal to the difference between the maximum and the minimum of convergence. Landolt's device consists of a blackened metallic cylinder which is to be fitted on to a candle. The side of the cylinder turned toward the eye presents a narrow slit (0.3 mm.), which is illuminated

by the flame of the candle and serves as a fixation object. Beneath is attached one end of a spring tape-measure, graduated on one side in centimeters and on the other in the corresponding numbers of meter-angles (or what amounts to the same thing, in diopters). To find the punctum proximum of convergence, the case holding the tape-measure is held beside one of the eyes and the cylinder drawn about two-thirds of a meter away from it, directly in front of both eyes, with the illuminated slit turned toward the patient, who then fixes on the light streak while it is moved directly in the median line closer and closer to the eyes. The moment the streak of light begins to broaden or double, the point of greatest possible accurate convergence has been reached, when one side of the tape will indicate in centimeters the distance of the punctum proximum of convergence, and the other side the corresponding maximum of convergence in meter-angles; for instance, a convergence nearpoint of 11 c.c. corresponds to 9 meter-angles. In the mentally dull, the examination can be made much easier by having a colored glass before one eye so that doubling of the light streak will be recognized the instant it occurs. A much simpler method that we have used for 6 to 8 years past, is to employ the small electric light used in making the Maddox rod test at the reading distance. If in a darkened room this small light is held 13 inches from the eyes of the patient a tiny bright image of it is seen in the center of each cornea. As the light is carried toward the eyes and they follow it in, the images also move inward toward the nose; but when the light has been approximated to within about 4 inches of the root of the nose, one or both eyes of the average patient will refuse to further converge and the bright corneal image will be seen to roll outward. The moment the break occurs marks the near point of convergence. For instance, if one eye breaks away from the convergence act at 5 inches this is the convergence near point (C. N. P.) and expressed in meter angles would equal 8 meter angles. A 3-inch convergence near point would equal 13 meter angles (M. A.), and a 2-inch convergence near point would equal 20 meter angles of convergence.

The minimum of convergence (or convergence far point) is determined by the following method: Make the patient fix the flame of a candle at a distance of 6 meters or more and find the strongest prism *base in* before one eye that can be overcome without causing homonymous diplopia, then divide the number of the resultant prism by 7, to obtain *approximately* in meter-angles the amount of deviation of each eye. Thus, if abduction at 20 feet is 7 degrees the minimum of convergence is 1.00 meter-angle; if 8 degrees it is 1.14 meter-angles; and if 6 degrees it is 0.85 meter-angle.

In the normal state the average maximum of convergence (p) is about 10.5 meter-angles (about 3 1/2 inches as the convergence near point) and the average minimum of convergence (r), about 1 meter-angle; therefore the mean amplitude of convergence (a), as expressed by Landolt[1] would be, $a = 10.5 - (-1) = 11.5$ m. a., or if the maximum be 12.5 meter-angles and the minimum 1.5 meter-angles the amplitude of convergence would be 14 meter-angles. The calculation is one of simple addition and subtraction, and it will be observed that it takes account not only of convergence, but that it also reckons the extreme limit of divergence (as found with prisms) as the measure of the *minimum of convergence*, a fact that the student will do well to ponder and settle for himself before proceeding to the study of any practical office-problems in anomalies of convergence and divergence. As above stated, the two acts are so intimately bound up that it is relatively impossible to dissociate them, and investigation of one side of the question necessarily involves the other.

Careful weighing of all the facts connected with musculodynamics seems to warrant the following tentative conclusions:

1. That the degree of adduction (prism convergence) for 6 meters given by most writers as proper in the first office-examination cannot be reached by healthy eyes except after practice in prism convergence.

2. That primary adduction at the first office-examination will often not exceed 20 degrees.

[1] The Refraction and Accommodation of the Eye, 1886.

3. That adduction (prism convergence) can often be quickly trained to 50 degrees and not infrequently to 75 degrees, and in some cases even to 90 or 100 degrees.

4. That estimation of the *amplitude of convergence* is the best test of the real power of convergence and its limits.

5. That abduction as found at the first office-examination is fairly constant, and in orthophoria will rarely fall below 7 degrees.

That the ratio between adducton (prism convergence) and abduction (prism divergence) for 20 ft., ranges from 8 to 2 1/2:1 No arbitrary standard can be fixed as yet, simply because the figures thus far offered have been largely a matter of personal equation.

MEASUREMENT OF ARC ROTATIONS.

It is well to supplement prism rotations by the estimate of the arc rotations in all hyperphorias of any degree and in all lateral deviations of an obscure nature. The hand perimeter of Schweigger is well adapted to this purpose and is used as follows: The perimeter is held by the patient in the same position as for ordinary perimetry. A black strip on which a 2 or 3 letter word in ordinary print is pasted is then started from the fixation point and moved as far up, down, in and out as the patient can carry the eye and still read the word without moving the head. These figures are noted and entered on an ordinary perimetric chart and when the points thus charted are connected by lines a fair idea may be gotten of the arc rotations of the eye.

This method is really of value in many cases, but fails of necessity in presbyopes who would have to wear glasses during the test. The moment the test object or word passes beyond the degree marking the edge of the glass the test loses all value. The instrument applicable in all cases is that devised by Stevens known as the tropometer (Fig. 48). "It consists essentially of a telescope, in which an aerial image of the cornea is formed near the eyepiece. A scale, graduated to measure the rotations of the eye in all directions, is placed at the point where the image is formed." After placing the head in the head-rest, so that the glabella and

the commissure of the upper lip are exactly vertical, the patient fixes on the center of the object-glass. Under the direction of the surgeon, the patient turns the eye under examination to the fullest extent in the four principal meridians, while the head is kept absolutely immovable. The effect registered on the scale by

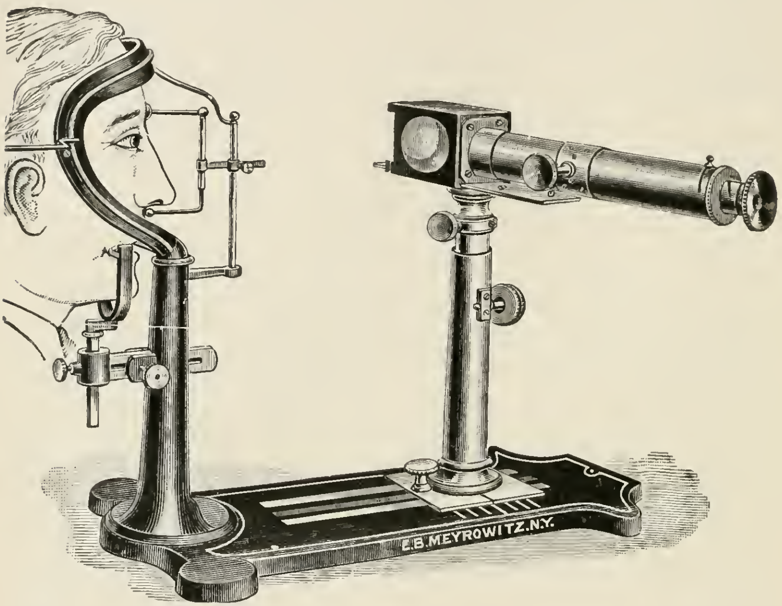


FIG. 48—The tropometer (*Annales d'Oculistique*, English edition, July, 1895).

movement of the cornea is shown in Fig. 49. These rotations are given approximately as follows:

Upward	33 degrees (of arc).
Downward	50 degrees (of arc).
Inward	48 to 53 degrees (of arc).
Outward	48 to 53 degrees (of arc).

Our own figures for 100 eyes with normal muscular balance show the following average rotations with the tropometer:

Nasal 52	Temporal 50
Upward 32	Downward 56

They represent the four principal arc rotations. In all the degrees of the circle between them, the rotations will be accomplished by the combined action of the muscles normally turning the cornea to these positions, and when these points have been found and connected they map out the *monocular field of fixation.* This varies much however, according to different observers. The discrepancies arise from variations in the normal power of the muscles of one individual as compared with another, and the amount of attention and effort of which an individual is capable.

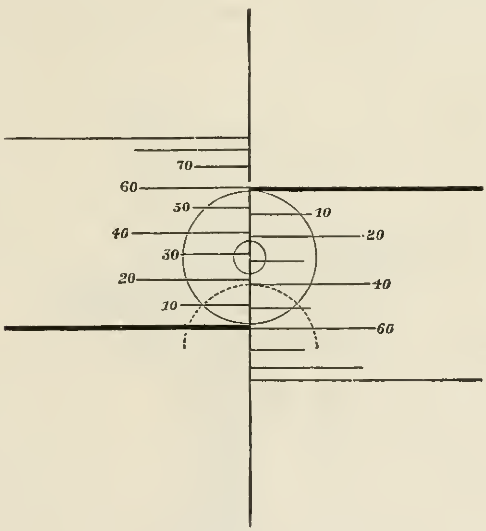


FIG. 49.—Scale for tropometer.

The *field of binocular fixation* comprises that part of the visual or form fields that is visible to both eyes at the same time without movement of the head, but with movement of the eyes. It is not necessarily identical with the field of binocular single vision, although the limits are practically the same. Near the periphery of the field of binocular fixation, the images of the object may not fall upon the yellow spot of each eye, but upon adjoining portions of the retina, when insuperable diplopia will be manifest. The field of binocular vision is smaller than the united

fields of each eye, and that of binocular single vision is still smaller. The limits of both may be readily determined by perimetric measurement, using one or two small white dots on a black background or black dots on a white background. Those dots on the periphery where the single dot doubles or blurs, represent the limits of the field of single vision. They are approximately:

Up......60 degrees (Pooley)........45 degrees (Duane).
Down.. 70 degrees (Pooley)........70 degrees (Duane).
Right and left 60 degrees (Pooley)..55 degrees (Duane).

In old age the rotations are lessened, and in determining them at any age, the form and position of the globe in the orbit and

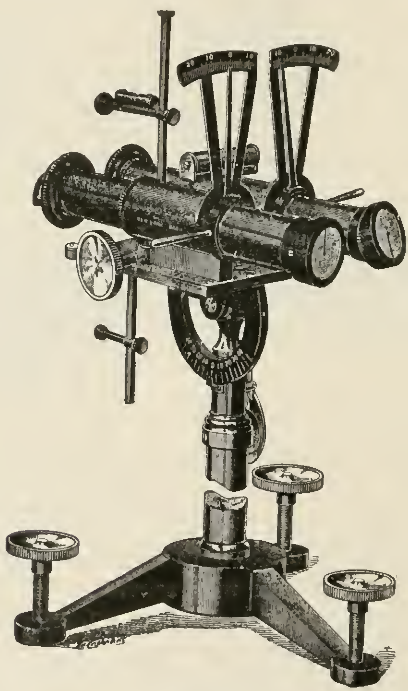


Fig. 50.

the orbital bones must be taken into consideration. Such wide variations as are shown in the averages given above are confusing

to one who endeavors to determine whether a certain movement is normal or otherwise. An excess of 33 degrees upward or of 55 degrees downward rotation should direct suspicion to the vertical muscles, and if, in the presence of esophoria or exophoria, the temporal and nasal rotations are normal, operation on the lateral muscles will scarcely be recommended and is justifiable only when the vertical muscles have been exonerated of causative influence by careful estimation of their rotations; this is especially important because high-grade lateral deviation tendencies are often an indication of unequal vertical tensions. It may be well to note, in passing, that the surgeon may learn by means of the tropometer the peculiarity of his own muscular status with respect to the horizon before proceeding to the use of the clinoscope (Fig. 50), also devised by Stevens, for accurate study of the horopter in all cases of anaphoria or anatropia, also cataphoria or catatropia.[1] In case the observer is exophoric or esophoric, the tubes may be adjusted in divergence or convergence, and in case of hyperphoria a correcting prism may be placed in a clip next the eye.[2]

[1] Anaphoria. Latent deviation of both visual axes above the horizontal plane of the head.

Cataphoria. Latent deviation of both visual axes below the horizontal plane of the head.

Anatropia. Manifest (apparent) deviation of both visual axes above the horizontal plane of the head.

Catatropia. Manifest (apparent) deviation of both visual axes below the horizontal plane of the head.

[2] Monocular diplopia is the result of irregular corneal or lenticular curvature, or of disease of the foveal region, and bears no relation to the subject under discussion.

ESOPHORIA.

Symptoms.—The symptoms of the tendency of the visual axes toward each other are local or ocular, and general or reflex. They vary according to the degree of the esophoria and the susceptibility of the patient. The local symptoms are in no wise distinctive. They are: Pain in the eyes and in their immediate neighborhood, flushing of the conjunctiva after use, photophobia, lacrimation, blurring of the print in reading, and inability to continue for any length of time the effort of looking at near or distant objects without becoming drowsy. As will be seen, the same symptoms may depend upon hyperopia or astigmatism, or any other muscular or refractive error. The reflex symptoms are exceedingly complex and unstable, and they vary from an occasional slight headache to intense migraine; from a simple restlessness to serious functional nerve-diseases, from a chronic insignificant disturbance of digestion to loss of appetite, interrupted digestion, and vomiting. A peculiarity of esophoria, not found among symptoms of refractive or other muscular anomalies, is that the patient often complains of seeing his nose, especially in close work. This symptom is largely imaginary, for the patient is no more inclined by esophoria to turn his eye in so far that coördination will be lost, than he is to have double vision for all objects. The complaints are not limited to the time of using the eyes, but often follow for many hours after a continued or unusual strain. Identical symptoms, although perhaps not so severe, arise from gazing intently in the effort to see clearly a distant object. Esophorics invariably are disturbed by traveling in the train, by looking at rapidly moving objects, by shopping, and after the theatre or opera. The symptoms are due, not directly to the internal muscles, but to the constant

effort under which the patient labors to prevent abnormal convergence. A tendency inward can be controlled only by an equivalent tension on the muscles of divergence. If the defect is so high that the external muscles cannot constantly maintain coördination of the visual axes, and one eye turns inward, the symptoms may be immediately relieved, but the patient will complain of diplopia. It is not always the highest degree of defect that gives rise to the severest symptoms. The disturbances above described never arise, for instance, in cases of manifest internal squint. It can be readily understood that the constant strain on the abductors to maintain parallelism of the visual axes may give rise, in certain subjects, to alarming reflex neuroses. No set of muscles, in any part of the body, can be in a state of constant contraction without making a deep impression on the nervous system. Indeed, hypnotic states are induced through an analogous process. The severity, then, of the symptoms must depend almost entirely upon the susceptibility of the individual to abnormal impressions, and the patients who suffer most in esophoria are those who habitually complain severely from apparently insignificant causes.

Etiology.—The causes that contribute to the abnormal tendency of the visual axes inward are local and constitutional. In many cases, it is quite impossible to satisfactorily explain the origin or the existence of esophoria. It may be stated, however, in general terms that it is found most frequently in persons of a neurotic disposition who have low grades of H. Ah or H. The susceptibility of the nervous system plays a most important rôle in the causation of muscular anomalies, particularly esophoria. By that expression is meant an abnormally acute impressionability of the nervous system induced by lack of proper nourishment from deficiency of oxygenated blood, a stasis of the venous or arterial supply from disturbance of the circulatory system—in short, a neurotic disposition. That an underlying predisposition to irregular nerve-activity is an essential contributing factor in the development of the muscular imbalance and the symptoms accruing from it is demonstrated by the well-known fact that many

individuals, who have the theoretic local conditions assumed to give rise to muscular asthenopia, use their eyes constantly without discomfort; furthermore, in the absence of the local causes that are conceded to produce esophoria, and in the presence of those that are usually causative of exophoria, esophoria is often present and gives rise to no symptoms. Identical inconsistencies are illustrated in the study of the etiology of other than eye diseases. Two persons exposed to the same influences will be seized with different affections or one will entirely escape. It is not strange, therefore, that we are often at a loss to ascribe the mysterious unbalancing of the ocular muscles to a competent cause, and are compelled to invoke the aid of the supposition of a deranged nervous organization.

The interdependent, constant, tenacious relation between the intra- and extra-ocular muscles furnishes an explanation of the existence of esophoria in a majority of cases. In health there is a certain related range of accommodation and convergence; every contraction of the ciliary muscle is accompanied by contraction of the adductors, or stimulus to the accommodation means stimulus to convergence. In emmetropia the meter-angle of convergence is determined by the diopters of accommodation. Thus, accommodation of 3 diopters induces convergence of 3 meter-angles. Now if, for any reason, an abnormal amount of accommodation is required to read at the ordinary near point (thus overstepping the normal range) a correspondingly increased demand on convergence is made, and a tendency to converge the visual axes to a point within the desired reading distance results. When, notwithstanding this unbalanced relation, the normal convergence of the visual axes is maintained, it can only be at the expense of the relative accommodation and convergence, and in any case it entails an unusual resistance on the part of divergence. If the range of the relative accommodation and convergence is not exceeded in this effort to secure binocular fixation at the reading distance, there will be no esophoria and no symptoms, and this probably explains the exemption of certain individuals. The refractive conditions that most frequently give rise to a disruption

of the relative range are hyperopia, hyperopic astigmatism (simple and compound) and low myopic astigmatism. To overcome hyperopic states of the refraction and thus secure good acuity of vision for both distance and near, unusual demand is made on the ciliary muscles, which in turn stimulates all the other muscles supplied by the 3rd nerve. Hence, an inward tendency of the visual axes is originated, but this, in the interest of binocular single vision, is suppressed and the condition remains a tendency only. The same reasoning applies to hyperopic astigmatism; myopic astigmatism of low grade is also a frequent and potent cause of ciliary spasm, and thus may be classed among the causes of esophoria.

Treatment.—When, by means of one or all the tests described on page 88, inward tendency of the visual axes has been repeatedly and conclusively determined, we are confronted with the practical problem of relief. The method selected depends upon 1, the severity of the symptoms, and 2, the degree of the esophoria. We recommend the adoption of the following course:

First. Use of a Proper Correction.—In all cases the estimation of any optical defect under complete paralysis of accommodation and the wearing, for some weeks, of *as nearly a full correction as can be borne with comfort.* This is essential. No other treatment directed to the restoration of the lost equilibrium of the muscles should be inaugurated until glasses correcting the ametropia have been worn sufficiently long to remove any pernicious influence born of constantly over-taxed accommodation. The experience of all ophthalmologists confirms this statement, and no surgeon of judgment or ability will apply treatment directly to the muscles until he has given the patient the opportunity to be cured by wearing glasses. Any co-existing general disturbance must also be relieved by appropriate medication. Diet should be regulated, exercise in the open air instituted, the time devoted to reading restricted; in short, every means tending to diminution of the unwonted excitability of the nervous system, or the derangement of any of the vital functions, must be employed.

Second. Convergence and Accommodation Repression.—Inas-

much as excessive accommodative effort is a chief agent in the production of annoying esophoria, it is well to try to remedy the abnormal convergence effort by quieting the overactive accommodation if possible. Should the use of a proper correcting glass prove insufficient to allay the patient's symptoms, resort may be had to the use of a plus 2 or 3 diopter spherical lens to be used in addition to the regular correction by means of a hook front or other device. With this the patient will read or sew for 15 minutes to half-an-hour, two, three or four times a day for two to four weeks. Complaint will of course be made that the reading or sewing must be brought very close to the eyes, but if the patient is reassured he will generally persist. Some drift readily into the practice, some with difficulty and much persuasion, and some never. It will seldom be necessary to resort to it save in esophoria of 6 degrees or more. In some cases the results are peculiarly gratifying and in others are a total failure.

Third. Prism Exercise and Nerve Sedatives.—By means of frequent and interrupted use of prisms, we endeavor to strengthen the power and increase the range of divergence, and unless there is actual preponderance of convergence, some cases of recent standing may be effectually treated by this method. Abduction normally equals 7 or 8 degrees when tested with a candle at 20 feet. In esophoria, abduction sometimes falls to 1, 2, or 3 degrees, and the *normal* ratio between abduction and adduction (about 1 to 3 or 4) is lost. A prism of 6 degrees, axis horizontal, is placed *base in* before the patient's eye, and the patient is instructed to look at the candle-flame 20 feet distant. If his abduction equals 3 degrees, the prism will produce insuperable homonymous diplopia; but if he advance to within a few feet of the candle flame, abduction increases as he advances, and he will reach a point where the two lights will fuse. He now recedes from the candle, maintaining its image single as long as possible. When the images have again separated he approaches the candle until the lights are fused, when he again recedes. This may be repeated a number of times, but no longer than five minutes at one sitting. On the second trial he will find that the

lights fuse easier than at the first. At the third trial it will be still easier, and in the course of a few days he will be able to fuse the lights at 20 feet. This exercise should be continued until asthenopic symptoms have disappeared for some weeks. The prism may be placed before either eye, or before one and then the other, and the effect will be the same. Fusion of the lights is not the result of contraction of one or the other externus alone, but is accomplished by forced action of all the muscles which participate in divergence—an extremely complicated function. (See Weiland's article in *Arch. of Ophthal.*, January, 1898.) In our experience, however, prism exercise has quite as often aggravated the symptoms as it has relieved them. A useful adjunct to the prism exercise is the internal administration of the tincture of belladonna, hyoscyamus, bromides, or some of the other sedatives, in as large doses as can be well borne. The effect of these remedies is to diminish the spasm or abnormal contraction of the adductors and benumb the sensitiveness of the nervous system.

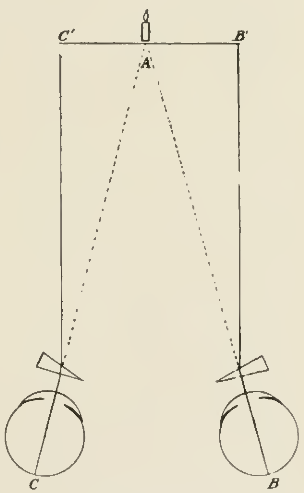


FIG. 51.—Effect of diverging prisms on excessive convergence.

Fourth. Correction by Prisms in the Position of Rest.—When, after appropriate trial by means of the above remedies and methods, it is felt that something must be done, and operation is out of the question, the symptoms may sometimes be relieved by the use of prisms, *bases out*, which correct a portion of the esophoria (Fig. 51). The degree of the defect requiring the use of prisms varies from 6 to 15 degrees. Defects under 6 degrees may frequently be corrected without them, while those higher than 10 degrees sometimes necessitate operation. The degree of the defect is determined in every instance by the result of testing at 20 feet. A constant relation does not exist between the convergence

for distance and for the near point in cases where equilibrium has been lost. For instance, a defect of 7 or 8 degrees for distance may be the same, or less, or more, at the near point, depending upon various concomitant conditions. The strength of prism should be much less than the total amount of defect. It is well for the novice to feel his way and not order more than 1 degree of prism *base out* in each eye as an experiment. It is a question whether wearing a prism correction does not develop an increase in the error; the very principle of its action forbids a cure. Under such treatment the tendency of esophoria is to increase, but whether this increase is due directly to the effect of wearing the prisms or to the unmasking of a latent defect is uncertain. Should the general health be improved by medication, change of environment, or occupation, etc., the defect may become less, but not often can this improvement be ascribed to the prisms. It is oftener the case that stronger and stronger prisms will be required to relieve the symptoms, and occasionally the case comes to operation. Our plan is to allow the patient to wear his prism correction without change as long as he is comfortable, permitting him to decide whether it shall be worn constantly or only for near work. In most instances, constant wearing only will give relief. It must be remembered that esophoric patients do not complain of headache or other symptoms consequent upon prolonged use of the eyes at the near point only, but intense gazing at objects in the distance, such as the preacher in his pulpit, looking steadily from a car window, or gazing at objects that are themselves moving, will give rise to these symptoms. The directions to the patients should be modified according to their occupation and symptoms, allowing them to judge as to the occasions when it will be necessary to wear the glasses. The following case is in point:

F. E., aged ten, is referred by his family's general medical adviser, who has exhausted his resources in endeavoring to quiet the boy's blepharospasm and general nervous irritability. Circumcision had been done and adenoids removed in hope of benefiting him, but without avail. The child was undersized for his age and the son of a neuropathic father and a gela-

tinous mother. His vision equalled 5/5 in each eye; his accommodation was normal. Esophoria of 10 degrees was present and no hyperphoria. Under atropin refraction was found to be +2.00 sph. in each eye. Because of his esophoria + 1.75 sph. was ordered for constant wear, but the child absolutely refused to wear the glasses because of blurred distant vision; +1.25 sph. was therefore ordered and worn with some relief to the symptoms, but six months later at the end of the school year the blepharospasm was as bad as ever. Convergence and accommodation repression were then tried, but as the family offered no coöperation it was finally decided to combine a 1 degree prism *base out* in each glass with the + 1.25 sph. In four weeks the blepharospasm had entirely disappeared and in four years has not reappeared. Today, after four years, he wears a 2 degree prism *base out* with +1.50 sph. in each eye. His total esophoria is 12 degrees, an increase of but 2 degrees in four years and the boy is growing finely and does his school work without any complaint whatever. It is quite possible that after he passes through puberty, the prisms may be taken out of his glasses and accommodation repression be invoked to allay any symptoms he may develop. Prisms in the position of rest give relief because, while worn, the patient's eyes are allowed to assume a position which is one of rest for them, and objects are held single by the refracting power of the prisms. In esophoria the visual axes have become converged, and prisms, *bases out*, bend the rays proceeding from the object outward toward the new position occupied by the fovea centrales. Here the position of rest is not parallelism, but convergence. The tendency to turn has become an appreciable turning. Prisms must be regarded as crutches that will permit the patient to travel comfortably over his rough road, rather than as a means of final cure.

Fifth. Operation.—In all cases operation should be reserved as the last resort. In low grades of esophoria—in fact, in all grades of esophoria not bordering on esotropia, the means recommended under paragraphs 1, 2, 3, and 4 should be given a proper trial and cannot be too much emphasized. Some writers go

even farther than this and state that no benefit whatever is derived from operation. Our experience does not justify this statement. We are convinced that many cases are operated upon that could be relieved by less radical measures, and, on the other hand, we have operated where other means have been tried and failed and have been entirely successful. The measure of success is the cessation of the symptoms and the restoration to equilibrium of the muscles. The condition known as equilibrium varies, as has been before noted, from esophoria of 2 degrees for 20 feet, to exophoria of 2 degrees for the reading distance. But it is our opinion that equilibrium or orthophoria is present only when, by testing with the Maddox rod, the streak of light passes directly through the center of the flame horizontally and vertically. Having decided that surgical procedure is necessary, we have the choice of advancement of the external recti or tenotomy of the interni. According to Landolt, tenotomy is never in place—advancement is the only surgical means. He bases this opinion upon the assumption that, in all cases, "the defect arises from a *loss* of divergence and not an *increase* or *spasm* of convergence; hence a weakening of the muscles which are not too strong is wrong in principle." If esophoria depends on hyperopia or hyperopic astigmatism—in other words, if the abnormal convergence is a reflex from unusual activity of the accommodation, the tendency of the visual axes inward is not a result of an insufficiency of divergence, but a physiologic sequence to inordinate action of the ciliary muscle, producing excessive convergence. Hence, in esophoria, tenotomy of the interni is more often productive of good results than advancement of the externi. Moreover, adjustment of the ocular axes is much more precise with tenotomy than with advancement. All such operations should be done under local anesthesia, so that the patient may be brought to the sitting posture and the final result controlled by some one of the tests alluded to.

It is not practicable to state in precise terms the exact degree of esophoria that demands or justifies tenotomy or to make an arbitrary law, any more than it is reasonable to say that in hypermetropia a certain number of diopters or fractions of a diopter

must be deducted from the full correction. The decision as to operation depends upon the effect of the methods of treatment previously outlined and on the severity of the symptoms. If we find that under accommodation repression, prism exercise and internal medication the defect remains unchanged or slightly improves, and the symptoms become less and less annoying, even though the degree of esophoria be moderately high, operation is not to be recommended. And, on the other hand, if a moderately low degree is associated with severe symptoms and only partial relief results from the wearing of prisms, operation is to be considered. In our own practice we are generally guided by the severity of the symptoms. *After other means have been faithfully tried and failed*, esophoria of more than 10 degrees will at times demand surgical interference. Prisms of more than 4 degrees in each eye cannot, as a rule, be comfortably worn. Moreover, prisms are changeless and constant in their refraction, while the muscular conditions of the eyes are changing and inconstant. We are attempting to correct a live physiologic function by means of a dead, unalterable piece of glass. The degree of esophoria varies as above stated—in fact, different estimates may be reached by different observers at the same examination, therefore, allowance for this variance must be made in the prism correction. After operation and consequent reduction of the degree of esophoria the conditions are often more amenable to prism treatment.

Should the operation be limited to one eye or should both be included? We are dealing either with excessive convergence or defective divergence, and in either case we have to do with more than one muscle and more than one eye. Theoretically, therefore, the operation should be divided between the two eyes, unless we have reason to believe that the esophoria is the result of an organic muscular change, limited to one eye, or is due to a nebula of the cornea, to high-grade optical defect, to vitreous or lenticular opacity, or to some impairment of vision, indicating that the esophoria proceeds from a local cause and is not the result of disturbance of innervation. Heterophoria is rarely met with in cases where organic imperfections of one eye lead to

manifest squint, and usually to amblyopia of high degree, and may therefore be excluded from consideration in this chapter. We are concerned with patients who suffer from no incurable affection of vision, have no organic lesion that can be determined, and who have, as a rule, fair acuity of vision in both eyes. We cannot, therefore, admit that the tendency of the visual lines inward is a monocular affection and that its treatment is consequently monocular. In all cases both eyes should be made to share the treatment as nearly as possible. Where double operation does not seem to be indicated, the symptoms can be relieved by the other means suggested without operation. It is a debatable point whether both eyes should be operated on at the same sitting or the second operation follow several days after the first. The points in favor of the former are, that under antisepsis the surgical procedure is free from danger; the incision is extremely small, involving but few tissues and these are superficial; the pain is insignificant and confined to the dragging forward (either by the hook or forceps) of the tendon and the cutting of its fibers; hemorrhage is unworthy of mention, the subsequent discomfort is little if any more after the double operation, the rotation of each ball is similarly and contemporaneously influenced; and finally the patient is gratified that the performance is completed. The advantages of the single operation are: less immediately subsequent annoyance; and the possibility that it will suffice for a cure. But such advantages are not worthy of serious consideration when compared with those of the dual operation. The progress of the operation must be marked by measurement of effect according to the change in position of the images of the candle-flame at 20 feet or of the streak if the Maddox rod is used. As often as necessary, the operation must be suspended and the result of the section of a few fibers determined, otherwise the operator is in the dark and his result is

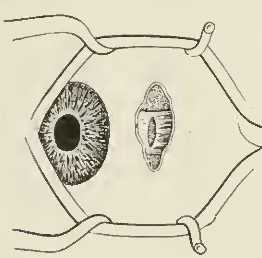


FIG. 52.

only approximate. In performing this operation it is well after dividing the conjunctiva to pick up the tendon of the internus with a forceps instead of a strabismus hook and simply button-hole it (Fig. 52). Further cautious divisions may be made above and below according to the indications furnished by the Maddox rod.

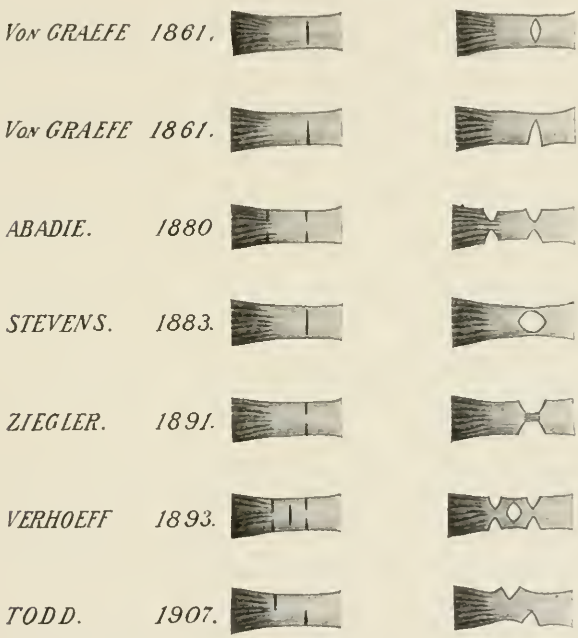


FIG. 53.—Various methods of doing partial tenotomy. (Ziegler, Annals of Ophthalmology, April, 1911.)

In this way every fraction of result secured from the division of even a few fibers may be most accurately studied, and overeffect avoided. In Fig. 53 various methods of doing partial tenotomy are portrayed. Our own preference is for the method of Stevens, although that of Ziegler has much to commend it.

EXOPHORIA.

For a long time it was held that exophoria was a purely passive condition resulting from diminution of the convergence impulse, but this conception must now be modified. That a certain percentage of all cases of exophoria does represent a passive or under action phenomenon (in reality an accommodative exophoria) is a thoroughly established fact. On the other hand, in a series of 441 cases of exophoria[1] it was shown that in 72 per cent. of the cases there was an associated H+Ah refractive status; and inasmuch as all hypermetropic conditions call for excessive accommodation impulse (hence associated excessive convergence) some factors other than subnormal accommodative impulse must be invoked to explain this association. Moreover, the term exophoria (like esophoria) while of great clinical convenience, is not altogether sufficient to the purpose for which it is employed, in that it gives no clew as to whether convergence be deficient, divergence excessive, or both divergence and convergence deficient.

For purposes of study, therefore, exophoria may be arranged in four classes:

1. Exophoria with subnormal accommodative power (generally allied with an M+Am refractive status, the old classic convergence insufficiency of Donders, von Græfe and Landolt).

2. Exophoria (in the presence of H +Ah) with normal divergence and deficient convergence.

3. Exophoria (in the presence of H+Ah) with normal convergence and divergence excess.

4. Exophoria (in the presence of H+Ah) with convergence and divergence both deficient.

Concerning exophoria with subnormal accommodative power it may be said that the cause is a myopic error of refraction. In

[1] Journal of the American Medical Association, 1906.

myopia and myopic astigmatism of two or more diopters, the demand on the accommodation, when reading or otherwise using the eye at the usual near point, is less than in lower errors of refraction, since the lens, already too convex to bring parallel rays of light to a point on the retina, is adapted for a distance inside of infinity, and the higher the myopia, the nearer to the eye is its far point, therefore the less need for contraction of the ciliary muscle. The muscles of convergence, deprived of the impulse associated with ciliary contraction, are handicapped and respond to the impulse to convergence only, instead of the impulse to convergence and accommodation. When we consider that vision at the reading distance is demanded by the majority of the human family hundreds of times daily, the final effect of such an insufficient innervation may be readily appreciated. After months or years a distinct loss of impulse results and exophoria appears. According to this reasoning the degree of exophoria has a distinct ratio to the grade of myopia, and, other things being equal, this is the true state of the case. Indeed, it is rare to find equilibrium of the ocular muscles and still rarer to find esophoria among myopes, for the same reason that esophoria is common among hyperopes.

Symptoms.—Patients with exophoria are quite likely to be neurasthenic to a greater or less degree, so that their description of all their symptoms is subject to a certain discount.

Headache.—Naturally this is the predominant symptom, especially when it persists after wearing a good correction. An analysis of the headaches complained of by 173 exophorics shows that the frontal type was present in 102, the occipital type in 60 and the temporal type in 11. These headaches are more or less constant, exist independently of use of the eyes for near work, and are exaggerated by near work, also anything which calls for much quick adjustment of the eye muscles, such as railway and trolley journeys, moving in a crowd, and shopping. On the other hand, anything which calls for prolonged steady fixation, such as attendance at the theatre or gazing fixedly at the preacher in his pulpit, produces the same effect. Particularly

trying to such patients is an expedition to a picture gallery, as the gaze is generally directed upward and exophoria has a tendency to become greater as the eyes are turned upward.

Of 200 exophorics in private practice, 140 gave testimony as to more or less vertigo, especially after near work. This particular vertigo may not be greatly annoying as it is of the most transient character, but the patient so seldom associates the symptoms with their ocular status that it will be elicited only by questioning. Ocular fatigue was present in 153 of the 200 cases, ocular tenderness in 98, conjunctival irritation in 121, and low grade photophobia (or glarophobia) was in evidence in 130. Nausea of a transient kind seemed fairly well related to the exophoria seventy-two times and drowsiness after near work, especially at night, was almost a constant symptom. In twenty-three cases gastric reflexes were admitted. By this is meant a nameless vacant sensation in the epigastric region. That a certain percentage of exophorics (who overcome their exophoria) have this reflex is indisputable. Two cases presented intractable superior dental neuralgia (with teeth in perfect condition) that yielded to treatment for the associated exophoria. It will, therefore, be seen that the symptoms are variable indeed, and just as with an uncorrected refractive error, they may be direct or remote, and may be almost individual or may appear in groups.

Diagnosis. *Diplopia Tests.*—A prism of 8 degrees is placed base down before the right eye, the patient gazing at a candle flame 20 feet distant. The upper image is the false one and belongs to the right eye. It is not directly above the true image, but is above and to the left. The prism *base in* before either eye that brings the two images directly in a vertical line is the measure of the exophoria. The phorometer of Stevens is a much readier application of the same principle, and by rotation of its lever the character and amount of the exophoria are shown at once.

Cobalt Glass Test.—In high-grade exophoria, diplopia will be produced by holding a cobalt blue glass in front of one eye, the image seen by which will be blue-red and smaller than that seen by the uncovered eye; the cobalt image will lie on the side opposite

to the eye before which the glass is placed, the separation depending on the grade of exophoria, expressed in terms of the prism, *base in*, necessary to fuse the cobalt and the true image. Defects of less than 3 degrees are, as a rule, not uncovered by this test.

Distorting Tests.—Of most value is the Maddox multiple rod. If it is so placed before the right eye in the trial frame that a vertical streak appears before that eye, the streak will fall to the left of the candle flame; and to the right if the rod is placed before the left eye; that is, the images are crossed. The prism *base in* before either eye that will displace or carry the streak directly through the flame represents the degree of the defect.

Cover and Parallax Test.—The patient gazes at a small brilliant object 20 *or more feet distant.* The eyes are screened alternately, thus breaking up fusion. As the cover or screen is carried back and forth from one to the other eye, the uncovered eye will move *in* toward the median line because while under the cover it has rolled out into the position most restful for it. The prism *base in* before either eye that will arrest the movement of the eyes is the measure of the exophoria. This test is wholly an objective one and its objectivity and accuracy give it great value. It does not consume more than one minute in its application and should never be omitted from the study of any case of exophoria. If it is desired to make it subjective by asking the patient after a few movements with the card whether the fixation object appears to jump from side to side as the card is shifted this is readily done, and so carried out becomes the parallax test. The prism *base in* before either eye that arrests the apparent jump of the fixation object is the measure of the deviation. This test is of the greatest delicacy, one-fourth of a degree of deviation being easily possible of detection in intelligent patients.

Tests for the Reading Distance.—In no class of cases will tests at the reading distance be more serviceable than in exophoria. For this purpose we have discarded the dot and line test, and use almost entirely the multiple Maddox rod and a small one candle electric light. The patient holds the light at the ordinary reading distance (about 13 inches) and the state of the muscles at

that point is estimated, the patient's correction invariably being worn during the test. In presbyopes the reading correction should be used when the test is applied or the observer may be led into error. Schild's, Baer's or any other modification of the apparatus just described may be used.

Too much stress cannot be laid on this test in exophoria, as is illustrated by the following fairly typical case:

J. G., thirty-five, accountant, complains much of ocular distress after working two to three hours consecutively over accounts. He had been carefully refracted three months previously and ordered a suitable correction. At that time exophoria of 1 degree for infinity had been noted. Estimation of the muscle status at his working distance revealed exophoria of 11 degrees and no hyperphoria. The convergence near point was 5 inches (12 1/2 centimeters). The abduction was 8 degrees and the adduction 12 degrees. He was given convergence training which improved the condition somewhat. Finally a 1 degree prism *base in* each eye was incorporated in his regular correction and this prismatic glass ordered *for his work at accounts only.* Within four weeks all his ocular distress had disappeared.

Supplementary Tests. *Prism Convergence.*—As has been noted in a previous chapter, prism convergence is such a variable factor that it may prove misleading if too much reliance is placed upon it. Yet it will often elicit some interesting information and should be estimated in all obscure cases. In general terms there should be at least twice if not three times as much prism convergence as prism divergence in all but high degrees of exophoria.

Prism Divergence.—Prism divergence is commonly a comparatively fixed quantity and if found to be 6 or 8 or 9 degrees at any one sitting it is fairly sure to measure the same at any future sitting. It, therefore, becomes a factor of marked value. Usually it is not found to be more than the normal 7 degrees.

The Convergence Near Point.—In every case of exophoria this element is of paramount importance. Landolt many years ago devised for the ready estimation of the convergence near point

his ophthalmodynamometer (see page 106) which is of much service. For the last eight years we have employed for the same purpose the tiny electric light of the De Zeng electric ophthalmoscope. With the room half darkened, and starting at 15 inches from the face, the patient watches the light attentively as it is carried in the median line closer and closer to the face. At about 10 inches from the eyes the bright small corneal images from the electric light become plainly visible to the surgeon and it is easy to watch the behavior of these images as the light is carried toward the root of the patient's nose until one or the other of the corneal images shows that the eye to which it belongs is no longer being converged. This is the near point of convergence and one can thus readily calculate the number of meter angles of positive convergence at the patient's disposal. For instance, if the eyes refuse to converge inside of 4 inches there are 10 meter angles of positive convergence; if they refuse at 5 inches there are 8 meter angles; if at 3 inches there are 13 meter angles. The test as thus applied is objective, while Landolt's proceeding is subjective and open to all the disadvantages of every subjective test; it is rapidly applied and of proven value. If we now divide the prism divergence (previously estimated) by 7 and add the quotient to the positive convergence we shall have the total amplitude of convergence. Usually the average patient has from 1 to 1 1/2 meter angles of negative convergence (prism divergence) hence the calculation becomes very easy. Thus: Fannie G, aged eighteen, shows 3 degrees of exophoria for distance and 15 degrees for the reading distance. Her prism divergence is 7 degrees (equals 1 meter angle of convergence) and her convergence near point equals 8 inches—that is, meter angles of positive convergence. Hence her total amplitude of convergence is but 5 positive and 1 negative or total 6 meter angles. Inasmuch as anyone who wishes to use his eyes much at the reading distance should have at least 11 meter angles of convergence amplitude (that is to say a convergence near point of at least 4 inches) it is needless to say that this young woman had great difficulty in using her eyes for any reading, writing or

sewing. In some cases of exophoria, the tropometer will be of value in determining whether both eyes share equally in the deviation (innervational exophoria) or whether one eye is markedly deficient in its adducting power as compared with the other (anatomic exophoria).

The four principal tests in the study of all exophorias are: a. the exophoria for infinity (or distance); b. the prism divergence; c. the exophoria at the reading distance, and d. the convergence near point. There are many supplementary tests that may be used to advantage, but knowledge of the four facts just mentioned is essential for the proper treatment of any case of exophoria.

In all cases of exophoria the various tests should corroborate each other closely, in which case no difficulty should be experienced in arriving at the conclusion that the visual axes have a tendency away from each other. Here again, much as in esophoria, we are dealing with the relation between divergence and convergence and (with the exception of the anatomic cases) not with the externus or internus of one or both eyes, but with the diverging power of the externi and the obliques as opposed to the converging power of the interni, assisted by the vertical recti.

Finally, no test for exophoria is reliable that is instituted any time within twenty-four hours of the time that a mydriatic is used in the eyes for the same reasons set forth in the chapter on Esophoria. Tests should be made before the mydriatic is used, and if with the mydriatic effect in the eyes not until the mydriatic has been exerting a constant effect for two to three days.

Treatment.—The treatment of exophoria (or latent divergence) calls for much discrimination.

1. As most of such patients are more or less neurasthenic (as has been already pointed out), too much emphasis cannot be laid upon the value of right living. This implies moderation in all things (including diet, smoking, drinking and all other daily life habits). Hydrotherapy, either by means of warm baths before retiring or a quick cold sponge on arising—preferably the latter—is often of service. An additional hour or two of sleep will at times prove the saving measure.

2. *The Correction of the Error of Refraction.*—This reiteration may become wearisome, but the authors concede that the great proportion of muscular insufficiencies are directly or indirectly originated by ametropia, and believe they would be remiss in their duty unless they insisted on every opportune occasion that treatment should be invariably inaugurated by correction of any optical defect that may be present, for the double reason that the optical defect may be the sole source of the muscular symptoms and its correction may prove the cure of the same; further, no restoration to equilibrium can be expected when the cause thereof is acting. In exophoria associated with H+Ah it is the part of wisdom to undercorrect the spherical element of the refractive error by anywhere from +0.50 to + 1.00 sphere according to the degree of the deviation. This extra stimulus to the accommodation will serve a good purpose in evoking extra stimulus to the convergence, the thing most needed in latent divergence.

For the same reason we encounter the accommodative variety of exophoria in M+Am. The explanation is found in the incomplete or altogether neglected use of the accommodation. The myopic eye has a far point inside of infinity, its distance from the eye depending upon the diopters of myopia. Thus in M. of 3 diopters the far point is 13″. This is a convenient distance for reading and no accommodation is required. In M. of 5 diopters, the far point is 8″, which is too near for the exercise of accommodation. In such cases, therefore, convergence is deprived of the usual associated stimulus of the ciliary muscle, and gradually weakens. It can be readily seen, therefore, that in the presence of myopia the correction of the refraction is a necessary part of the treatment of the muscular anomaly. Indeed, when the myopia is not too high or the patient too old, it is often the only treatment demanded. The correction, in order to be effective, should be worn constantly.

If, after two months' use of proper correcting glasses muscular equilibrium is not restored or the symptoms persist, medicinal treatment may be resorted to.

3. *Nerve Tonics.*—The remedy that has been most warmly

endorsed and that the authors have found extremely serviceable is tincture of nux vomica in ascending doses. Strychnin, the alkaloid of nux vomica, is for some reason less efficient. The beginning dose of the tincture should be 20 drops thrice daily, increasing the dose one drop every day until 50 to 60 drops thrice daily are taken or until symptoms of physiologic action of the drug appear. Then the dose is similarly decreased day by day until the original dose is reached, when the drug may be withdrawn. The measure that is of most service in connection with the use of nux vomica is training of the convergence faculty by means of prisms.

4. *Convergence Training.*—This may be of various kinds. We resort mainly to two methods. First, converging exercises with a pencil point held at arms length; this is carried in toward the root of the nose until the patient sees double when the eyes are closed and the pencil carried back to arms length and the movement repeated up to ten to twenty times. Such an exercise period should be gone through two or three times a day. In this way the convergence near point can be brought nearer and nearer and the positive range of convergence much increased. The same exercise to the right and left is sometimes helpful.

The range and power of convergence can be distinctly increased by regular prism exercise. Prisms, bases out, have been used in various ways by different workers for many years for the exercise of the adduction; Risley, Michel, Savage, Noyes, Gould and others. (Acknowledgement is gratefully made by the senior author to his friend and preceptor, Dr. Wm. Thomson, until lately Emeritus Professor of Ophthalmology in the Jefferson Medical College, for instruction nearly thirty years ago in his office, in the use of prisms both as a method of stimulating the innervational force of the external ocular muscles and as a means of relief in muscular asthenopia). The battery of Noyes or Gould is a convenient arrangement of prisms and can be employed with benefit, although the costliness of such apparatus limits its use to the consulting room. For home use the prisms are to be mounted either in an ordinary spectacle frame or in one of the numerous frames

specially devised for carrying square prisms so that they may be increased ad libitum. (See Fig. 53a). The patient is given a pair of 5 degree prisms in the frame, with which he walks toward a candle flame placed at the opposite side of the room. The prisms are not looked through as he goes toward the light, but are raised. As soon as he approaches within 2 feet of the light the prisms are dropped before the eyes and he backs away from the light gazing steadily at it all the time. Should the light appear double any time while he is backing across the room, the patient immediately raises the prisms, walks toward the light again and backs away with the prisms before the eyes until he

FIG. 53a.—Training prisms for exophoria.

can go the whole length of the room without seeing the candle double. This is done from ten to fifteen times two or three times a day. After two weeks 10 degree prisms are ordered to be used in the same way and after another fortnight 15 degree or 20 degree prisms according as the patient learns rapidly or slowly the trick of dissociating his accommodation and convergence. There is a class of patients for whom convergence training as above described is particularly indicated; namely, the exophorics who present but 1 or 2 degrees of deviation for infinity with anywhere from 10 to 15 degrees of deviation at the reading distance (prism divergence being normal). These patients respond finely to convergence training as a rule. It will often require the strictest injunctions on the part of the surgeon to impress upon the patient the necessity for, and the importance of these calisthenics, and to bring him to realize that the partial or complete relief of his asthenopia is largely in his own hands. The coöperation of the patient once secured, the prisms can be rapidly increased in strength and the fusion force often carried to 100 degrees of prism inside of eight or twelve weeks, in which event the patient is

pretty sure to have experienced marked relief from the asthenopia. The main difficulty in this method lies in the persistence necessary to convince the patient that little is to be hoped for without the heartiest coöperation. Many cases of severe muscular asthenopia can be made quite comfortable by the combined action of increasing doses of nux vomica and prism exercise; indeed, their value has been universally acknowledged. An interesting clinical fact in connection with this method of treatment is that if a patient start with an exophoria of 10 degrees for distance and 18 degrees for 15 inches, (the occupation distance), the asthenopic symptoms may be entirely dissipated by nux vomica and prism exercise for two or three months, notwithstanding which the exophoria for distance will remain at 10 degrees, while that for near may have fallen to 3 degrees or 5 degrees, or have entirely disappeared; all of which goes to show that exercise of the adduction may increase the range of convergence (as can be found by prism tests of the adduction and abduction), but will seldom influence to any extent the muscle-balance for 20 feet. In other words, we have simply made easy to the patient a much-needed coördination, and have perhaps at the same time trained the cortical fusion centers to a higher degree of efficiency. These measures are peculiarly useful in individuals who have passed through a debilitating illness, such as typhoid fever, la grippe, measles or diphtheria; also in young and growing subjects, and their action is always enhanced by life in the open air, general muscular exercise and nutritious diet. Occasional cases will be met with whose symptoms are decidedly aggravated by prism training. This is frequently a clinical indication for the use of prisms permanently in the position of rest in the patient's glasses.

5. *Prism Correction.*—When the symptoms of exophoria are not relieved by the preceding measures and are of such a severity that relief can be obtained only by relaxation (or easing) of convergence, the wearing of prisms in the position of rest is to be considered (Fig. 54). The use of prisms, *bases in*, to be incorporated in the patient's distance correction has been deprecated by many

authorities who contend that exophoria for infinity increases under the constant wear of such prisms; furthermore, that they do not cure the deviation, but only relieve the symptoms. That this is true within certain limits must be admitted; and yet when correction of the ametropia has failed to relieve, and attempts at training the convergence have been poorly borne the trial of prisms *bases in* in an extra or hook front experimentally seems justifiable. In properly selected cases it will be found that when convergence has been made somewhat easier for the patient by the use of rest prisms, the convergence may then be trained to a very considerable point and the exophoria for infinity thus kept from increasing. This was our experience in seventeen out of forty-two cases in which prisms *bases in* were ordered for constant use. If the exophoria is the result of a temporary loss of convergence from overuse of the eyes or exhaustion of the nervous system the prisms not only give comfort, but they may gradually be reduced in strength and finally discarded. While the prisms are worn the visual axes diverge and the globes assume that position in the orbit most restful for them. The effort of convergence is partially in abeyance and the strain of opposing the power of divergence is relaxed. The eyeballs are diverged and single vision is maintained by the prisms. For these reasons it is good practice to allow a goodly proportion of the exophoria to remain uncorrected by prescribing prisms that permit constant but limited use of convergence. Thus, in exophoria of 8 degrees for infinity, prisms of 1 to 2 degrees before each eye may be tried. It is well to feel one's way with prisms, the purpose in view being not to supplant convergence, but simply to ease it up. Whether the prisms should

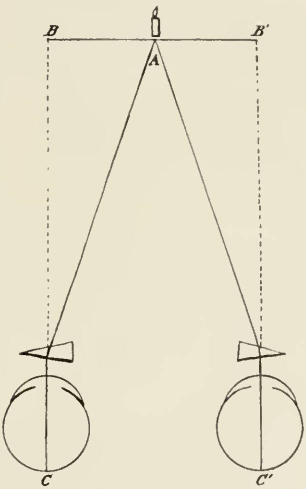


FIG. 54.—Effect of converging prisms in exophoria.

or should not be worn constantly depends upon the symptoms accompanying the use of the eyes for distance. Defects of 4 degrees and less for 20 feet rarely require their use. Higher defects will usually demand their constant use. There is a limit, however, to the benefit to be derived from prismatic correction according to sex, age, occupation, etc., but in general terms the highest degree of prism that can be comfortably worn may be placed at 5 degrees before each eye.

6. *Prism Correction for the Reading Distance.*—In certain cases of exophoria it is at times sufficient to employ prisms in connection with the reading or occupation glasses only. This is especially true in presbyopic exophorics. The influence of age in unmasking or perhaps even developing exophoria is unmistakable. Reference to the accompanying table

TABLE E.

Decade.	No. of times found.	Decade.	No. of times found.
Under 10	6	41 to 50	124
11 to 20	34	51 to 60	59
21 to 30	90	61 to 70	14
31 to 40	106	Over 70	6

setting forth an analysis of the incidence of exophoria in the various decades of life will show that the decade from forty to fifty presents the greatest number of cases of exophoria. Less than 1/10 of all the cases occurred prior to the 20th year of life and over one-half of all the cases in the period between thirty and fifty years of age. It is not at all uncommon to find presbyopes who reveal but 1 to 2 degrees of exophoria for infinity with 8 to 12 degrees for the reading distance. Prism training often produces the happiest results in such patients, but when it fails one need not hesitate to incorporate a 1 to 2 degree prism *base in* in each lens (preferably 1 degree) and the effect is generally most gratifying. Decentration of the lenses will accomplish the same result, but more will be said of this in another place.

When all the procedures mentioned fail to afford relief we are obliged to consider operation.

7. *Operation.*—The surgeon has the choice between tenotomy of the externi on the one hand and advancement (with or without resection) of the interni, or of the capsule, on the other hand. If exophoria depends upon lessened convergence power, there would seem to be no alternative between these two procedures; that the choice must fall upon measures that will strengthen convergence rather than weaken divergence. In our own practice tenotomy of the externi for exophoria is not often resorted to. Advancement is performed in nearly every instance. We believe this is correct practice founded upon sound physiologic principles, yet we are obliged to admit in all fairness that our results have not always been as satisfactory as we or our patients could wish. Advancement (capsular or capsulomuscular) is far more difficult and tedious to perform than tenotomy, more painful and incapacitating to the patient and it may not increase the power of convergence. When accurately done we believe it is superior to and gives more lasting results than tenotomy. On the other hand, tenotomy can be done under local anesthesia; it is extremely simple and easy to perform, it can be made most accurate, it inflicts but little pain, and a very short period of incapacity and when divergence is greatly in excess occasionally affords brilliant results. In patients under thirty years of age it would generally be best to resort to advancements; after that time, when the divergent forces are more in evidence, tenotomy may be considered when divergence can be plainly shown to be greatly in excess of normal. In deciding as to operative treatment, the etiologic relation between exophoria and hyperphoria must be constantly borne in mind. (See chapter on Vertical Imbalance and Lateral Imbalance). Whether one or both eyes shall be operated upon is to be decided on the merits of each case. The effect in degrees gained by a single operation depends largely on the preserved power of adduction. As a rule, it does not exceed 5 to 6 degrees and when more than this amount of correction is demanded, as is the case in most subjects for operation, each internus should be advanced. It is true that more effect in the divergence can be obtained by *full* tenotomy of the externi and at first sight this operation would

appear to be indicated, but *full* tenotomy of one or both externi should never be done because of the serious objection of diminished external rotation.

Moreover, as has been said, there is not so much promise of permanency of result and increase in adduction with tenotomy as with advancement. So that if the surgeon is sufficiently sure of his technique, he will probably obtain the best results by operating on both interni, dividing the amount of correction between the two eyes (as nearly as may be), at the same sitting. Postponement of the second operation is not contra-indicated, yet the preference should be given to the double operation since by this plan the dread and anxiety of the patient are reduced more than one-half and the eyes are in a condition to respond equally to the innervational impulse. If these facts are explained to the patient it is improbable that he will raise objections to the plan proposed, but will submit philosophically to the reasoning of the surgeon. (For description of tenotomy and advancement see chapter on Operations.)

HYPERPHORIA.

Hyperphoria is that condition in which there is a tendency of one visual line in a direction above that of the other. Hyperphoria (latent vertical squint) differs from hypertropia (manifest vertical squint) only in that single vision is not possible in the latter condition, and the eye that is more ametropic, or is at the greater muscular disadvantage, gives up the struggle and actually deviates upward or downward.

If we accept the above definition, it will be found that hyperphoria is by no means rare, nor is its presence inconsistent, in some cases, with perfect health and comfort in using the eyes, in which event there can be no excuse for treatment of the tendency to deviation.

An analysis of the findings at the first visit in 700 consecutive refraction cases occurring in our practice showed that 20 per cent. of all of them revealed hyperphoria of one-half degree or more; and a recent study of 3600 refraction cases in our private practice indicates 7 per cent. in whom prolonged study of the case revealed 1 degree or more of hyperphoria. Carpenter found it in 35 per cent. of his private cases at the first consultation, Posey in 13 per cent., Bannister (working among the U. S. Navy recruits) in 7 per cent., and Howe and Williams in 16 per cent. This would indicate that about one in ten of all patients who are the subjects of muscular or accommodative asthenopia will during the preliminary examination display some degree of vertical imbalance, either alone or associated with esophoria or exophoria. It should ever be borne in mind, however, that hyperphoria (like esophoria or exophoria) occurs in a fair percentage of individuals in whom careful refraction alone without any special optical treatment of the muscle status seems to give perfect relief. Some people carry a vertical error of 1 to 2 degrees all through a life of high tension and large usefulness without any particular discomfort, while in

others one-half to three-quarters of a degree is sufficient to make life a burden. Nature is marvelously elastic in her adjustments. So that the mere finding that a patient has hyperphoria is of itself nothing until the vertical deviation is viewed in its relation to his or her heredity, environment, temperament, occupation, physical condition and last, but most important, the refractive status of the eyes. Hyperphoria of 3 degrees in a soldier may easily prove a negligible quantity; while hyperphoria of one to one and a half degrees in a student, accountant, stenographer, private secretary, etc., may alter their whole career. One must be on the lookout always for spurious hyperphoria. The following case is in point:

A. W., female, aged sixteen. Comes on account of difficulty in all near work. General health fair. Vision R. E. 5/7; L. E. 5/9. Accommodation normal. Muscle status esophoria 4 degrees, left hyperphoria 2 degrees. Under atropia the refraction status was

R. +0.50 sph = +1.25 cyl. 75 degrees
L. +0.62 sph = +1.00 cyl. 135 degrees

after two weeks use of the cylinders only, the hyperphoria disappeared nor was there any in evidence 1 year later when the case was studied again. Such cases are not rare.

This class of cases (ametropic), along with those secondary to gout, rheumatism, central nervous lesions and other general diseases, comprise what are known as *temporary* hyperphorias in contradistinction to true or *permanent* hyperphorias which occur independently of constitutional conditions, and persist after long use of the closest correcting lenses.

Symptoms.—The symptoms of hyperphoria are ocular and reflex. The ocular symptoms are:

1. *Chronic hyperemia of the lids*, often giving rise to a condition aptly likened by one writer to the "hot eye" of gout.

2. *Epiphora*, generally unilateral and seemingly without nasal or ocular cause.

3. *Defective Vision.*—It is not unusual to find vision of but

2/3 to 1/2 in an hyperphoric eye even after accurate correction of a low-grade ametropia; indeed, it may be impossible to give each eye better vision than 5/7, a fact easy of explanation, however, if it is borne in mind that an actual vertical imbalance of 1 degree will result in a separation of images at 20 *inches* of 6 1/2 mm. Such a patient, if a worker at the occupation distance, will most surely be troubled from time to time.

4. *Confusion of images*, which is the result of the transient diplopia. It may be urged that many students carry a hyperphoria of 1 degree or more through a busy career extending over many years without experiencing any annoying symptoms whatever. Such persons either possess a physical and nervous system that would weather any storm, or they have, consciously or unconsciously, learned, by carrying the head toward one or the other shoulder, to neutralize their hyperphoria wholly or in part. This habit constitutes the fifth important symptom of true hyperphoria, namely:

5. *Carriage of the Head.*—The head is usually tilted toward the shoulder opposite to the hyperphoric eye, a statement that may sound strange, but when it is remembered that in right hyperphoria the image is really seen lower by the right eye, it naturally follows that the head must be tilted toward the left shoulder if the images are to be brought to a level and binocular vision thus rendered an unconscious act. Græfe referred to the turning of the face to one side in "insufficiency of the interni" to aid the weak adductors, but he said nothing of the peculiar carriage of the head in hyperphoria, which is equally common.

6. *A peculiar facial expression* will often be noticed, especially when an hyperphoric is in animated conversation; several furrows will ridge themselves above one eyebrow, and even the eyebrow itself may be raised above the level of its fellow from 4 to 10 mm., giving a quizzical expression to the face. There should also be mentioned, in this connection, the wide open eye, or stare, seen in many hyperphorics. Occasionally an apparent ptosis of many years' duration is dispelled by prismatic or operative correction of hyperphoria.

Not less important, as immediate symptoms of vertical deviation tendencies, are the painful eyes, photophobia and drowsiness induced by any long-continued near work, notwithstanding that the patient is wearing proper lenses.

The reflex symptoms of hyperphoria include more or less constant headaches, amounting sometimes to a migraine, nausea, vomiting, dizziness, and vertigo. In some instances the latter is so marked as to cause momentary unconsciousness and a symptom-complex that has more than once led to a diagnosis of epilepsy or even cerebral tumor. We believe it is the latter class of cases that are vaunted by many as instances of essential epilepsy partially or even entirely curable by eye-treatment alone.

The headaches of hyperphorics are almost invariably aggravated by near work; also by moving in a crowd or watching rapidly-moving objects (panorama headache of Bennett). Other hyperphorics will escape the headaches, but present, instead, frequently recurring nausea, vomiting and vertigo, the latter especially brought on by any continued looking up or down.

Etiology.—Intrinsic hyperphoria is commonly due to overaction on the part of one muscle (hyperkinesis) or underaction on the part of another (hypokinesis). It may also result from anomalies in the formation and relative position of the orbits or from peculiarities in the attachment, development, insertion or action of one or more muscles. Age is now by general consent admitted as one of the factors in the unveiling if not in the production of hyperphoria. In a series of cases reported by one of us in 1900, one-third were under thirty years of age and two-thirds over that age. (Compare with the same facts in exophoria). We have found intrinsic hyperphoria in a child of twelve, but this is unusual. There is no satisfactory explanation for the increase or perhaps unmasking of a latent hyperphoria. The parallel process of uncovering of latent hypermetropia by advancing years naturally suggests itself and may have much to do with this seeming actual increase in hyperphoria. Congenital paresis of one of the elevators or depressors—usually the superior recti, is a not infrequent cause of hyperphoria (or *hypo*phoria) as will be alluded to later on.

Diagnosis.—The assertion that hyperphoria exists in a given case ought not to be based on any one test, but demands corroboration by every test at our command. No problem in ophthalmology calls for greater accuracy and adjustment of instruments and close observation. When we say hyperphoria we use a clinically convenient term. We have not told whether one visual axis is abnormally high or the other abnormally low nor have we

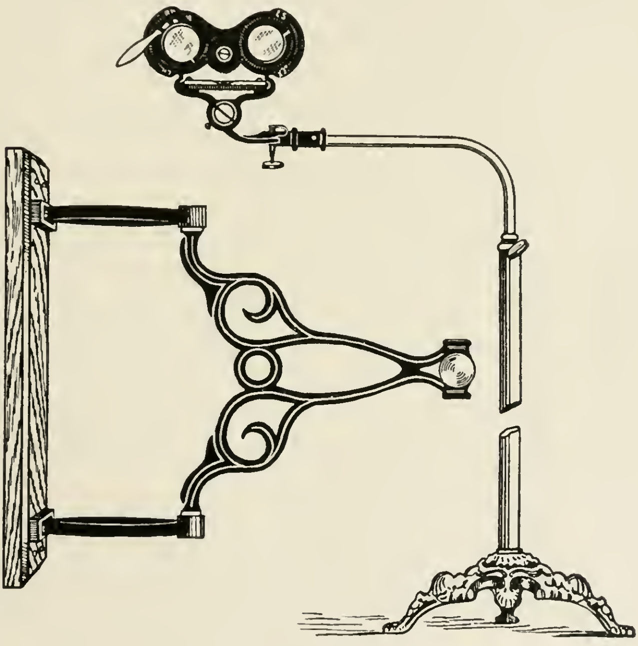


Fig. 55.—Reimold's optometer.

implicated any particular one of the elevators or depressors. So that after we have established the diagnosis of hyperphoria (or simply a deviation of one visual axis above or below the other) we should endeavor to discover if possible whether one axis has a tendency to become abnormally high or the other abnormally low.

In conducting many of the tests, Risley's, Reimold's or some similar combination of the optometer and phorometer (Fig. 55) gives satisfactory results.

Maddox Rod Test.—When the patient's face has been brought close up against the back surface of the optometer, and the latter been found to be absolutely level by means of the plummet or spirit level attached for the purpose, the compound Maddox rod is slipped into one of the cells, say the right one, and so turned that the streak of light is exactly horizontal. If the streak passes above the flame, there is shown either right *hypo*phoria (downward tendency of right eye), or left *hyper*phoria (upward tendency of left eye). If the streak be found below the flame there is right *hyper*phoria (upward tendency of the right eye) or left *hypo*phoria (downward tendency of the left eye). The same facts hold good when the Maddox rod is placed before the left eye, so that the test can be applied by placing the rod before either eye. The prism that carries the streak into the flame is the measure in degrees of the hyperphoria.

Some prefer to use the phorometer, but the Maddox rod has an added advantage in that if the hyperphoria appears to be of a different amount according as the Maddox rod is placed before one or the other eye, we thus learn that there must be a slight paresis of one of the elevators or depressors; that when the paretic eye fixes, the separation of the light and the streak will be greater (answering to the secondary deviation) than when the sound eye fixes. On the other hand, in some individuals the impulse to binocular vision is so overpowering a thing that they will frequently fuse the light and the streak, thus covering up their muscular error; but if the rod is quickly reversed several times in the trial frame from the vertical to the horizontal position one is much likelier to break up the strong fusion impulse in such a case and get at the true deviation.

Diplopia Tests.—If the phorometer be used, the prism or prisms with which diplopia is effected are already in position, and if one image be higher than the other, all that remains to be done is to rotate the prisms by means of the small lever until the images are level, when the resultant right or left hyperphoria will be indicated by the pointer on the arc graduated in degrees on the front of the phorometer, and can be readily read off.

Parallax Test.—Seat the patient 20 feet or farther from some small object that is situated directly in front of and on a level with the eyes. The object (a small luminous point of light in the middle of a large black area, for instance) must be so placed that it will not be projected upon any surface back of it. Note carefully that the patient's head is not tilted to one side, then carrying some sort of cover to and fro several times from one eye to the other, the patient is requested to watch closely whether the object as seen by one eye appears any higher than the other. If so, vertical imbalance is present and its degree is to be determined by the prism that will make the object appear on the same level with each eye. It will sometimes be necessary to carry the cover from one eye to the other fifteen to twenty times before any difference in level in the apparent position of the object becomes noticeable, but by this means the lower degrees of hyperphoria are frequently unmasked. The parallax test, when thoroughly understood, is susceptible of great delicacy of application and by it 1/2 degree of hyperphoria is easily detected. Duane, whom we must thank for the test, claims that he thus estimates even 1/4 degree of hyperphoria.

If the patient be ametropic, he must wear his correction during all the tests. Thus far we have determined only that one ocular axis has a tendency to deviate above, or the other below the axis of the fellow eye. We have not located the defect nor decided whether it be due to overaction of the muscles of one eye, or underaction of the corresponding muscles of the other eye. Resort must now be had to prisms, to learn the prism power of upward and downward rotation. This will be found to fluctuate from 2 to 3 degrees, but the fact of prime importance is that whatever the power of prism rotation, supraduction and infraduction should be equal. If the eyes can overcome a 3 degree prism, base down, they should likewise overcome a 3 degree prism, base up. A difference of 1 degree or more between the supraduction and infraduction of the same side indicates the probable existence of hyperphoria; moreover, a supraduction exceeding 3 degrees

(except in moderate and high myopia) is always suspicious and presumptive of hyperphoria.

If there be suspicion of hyperphoria and the supraduction and infraduction as found by prisms prove equal, it becomes necessary to map out the field of monocular fixation, especially the upward and downward limits. This is done by having the patient follow with the eye (but not with the head) a very small test-object, such as a black dot on a white card, which is carried as far up and as far down the arc of the perimeter as the eye will follow it without wavering or receiving a blurred image of the object. The arc through which the eye has rotated may be measured by the perimeter, the head resting on the chin-support and the fellow eye excluded as in ordinary perimetry. This examination should show at least 35 degrees to 38 degrees of upward, and from 50 to 55 degrees of downward rotation. For the precise measurement of these rotations, Stevens employs his tropometer (described above), and if upward rotation falls short of, or exceeds, 32 degrees, or if downward rotation exceeds or falls short of 55 degrees, the corresponding elevators or depressors are to be viewed as causative in the hyperphoria. If upward rotation is excessive, the hyperphoria is likely due to overaction on the part of the elevators of the hyperphoric eye. If downward rotation be excessive, the hypophoria is likewise due to excessive action (hyperkinesis) of the depressors of the hypophoric eye. If upward or downward rotation be insufficient (hypokinesis), the deviation is probably due to weakness, or even paresis of one or more of the vertical muscles, in which case diplopia can be readily elicited when the eyes (but not the head) are turned 20 degrees or more from the primary position, the diplopia increasing as the test-object is carried in the direction of the weak muscles.

Much information may sometimes be gained by search of the extreme upper periphery of the binocular fixation fields with a one candle electric light in a darkened room, a red or cobalt glass placed before one eye, to learn whether there is paresis of any of the elevators and similarly in the extreme lower field for paresis of any of the depressors. Hyperphoria and hypophoria

may be thus at times differentiated. For instance: V. M. M., female, married, twenty-three, referred by her physician on account of panorama headache, also train and trolley nausea and vertigo, so marked that she could not ride more than eight to ten blocks in the trolley cars. Use of her eyes for reading and sewing was limited to fifteen to twenty minutes, and this in spite of the fact that she was wearing a good correction. She was a tall, thin, poorly nourished girl, hyperesthetic and on the verge of neurasthenia. Both eyes were normal anteriorly and with her correction, which was:

R. +50+1.00 cyl. 100 degrees
L. +75+0.90 cyl. 80 degrees

vision was full and sharp 5/4 in each eye. The eye grounds showed the irritation and low grade congestion so often found with such muscular anomalies. Her muscle status was exophoria of 2 degrees for infinity and 5 degrees for the occupation distance. There was right hyperphoria of 8 degrees for infinity (when the eyes were in the primary position) and 9 degrees at the occupation distance. Search for diplopia in the extreme upper periphery of the binocular fixation field revealed vertical diplopia with the left image higher in the middle and left upper field, most marked in the left upper field where the separation of the images was about 4 inches. Paresis of the left superior rectus was thus plainly shown. On questioning her I learned that for years she had occasionally seen double when she looked at anything far up to the left, but thought nothing of it. So that the apparent diagnosis of *right hyperphoria* in the case immediately gave way to the proper one of *paretic left hypophoria.* It may be stated, in passing, that a 4 degree prism *base up* was incorporated in her left glass and after two years use of it she can ride 25 miles in a trolley car without the slightest ocular disturbance, she has lost all her headaches and hyperesthesia, and can use her eyes for one and one-half to two hours at near work comfortably.

Finally, when the tests for hyperphoria at 20 or more feet are concluded, search should be made for vertical imbalance at 15 to

20 inches. In this test Græfe's dot and line may be used or the dot only. Better yet is the small one candle electric light (suggested for the similar test in exophoria) to be held at 13 to 15 inches. Any vertical imbalance will then be recognized the moment the Maddox rod is placed before either eye and the amount readily measured. The patient should, of course, wear the proper correction (accurately centered) during this test and if presbyopic should wear that correction. It will frequently be found that hyperphoria for 15 inches is a trifle more than for distance.

The Relation Between Hyperphoria and Lateral Deviation Tendencies.—It has been claimed that many cases of esophoria and exophoria are directly dependent upon and caused by a tendency to upward deviation of one of the visual axes, and no doubt there is much truth in this claim. It is easy to conceive that when vertical tension is out of equilibrium the lateral muscles in their efforts to fuse images will tend to turn the eyes now in and now out, or will acquire a habit of forcing the lateral tendency in one or the other direction. This dependence has been illustrated by comparing the lateral muscles, in their efforts to fix, with the lifting of a log with a pair of tongs, the clamps of which are not in a horizontal line. Many cases have been reported where not only deviation tendencies, but *actual lateral turnings* have been cured by restoration of vertical equilibrium, and in the consideration of causes which lead to exophoria or esophoria, vertical tensions should be given a prominent place. Theoretically, all cases of uncorrected hyperopia ought to have upward and inward tendencies on account of the resultant action of all the external muscles supplied by the 3rd nerve, and the reverse will hold in exophoria, namely: a tendency down and out. This interdependence can be well shown when the grade of the defect is high enough to allow of diplopia with the cobalt glass test. It will be found by this test that there are few cases of lateral turnings that are not complicated with vertical turnings, and it will also be found that hyperphoria frequently changes from one eye to the other, according to the eye used in fixation. If the right eye should

fix and the left, armed with the cobalt glass, sees the false image to the left and below, we have hyperesophoria (really hypereso*tropia* while the cobalt glass is before the eye), and if the glass is transferred to the other eye, that eye now sees the false image and we have R. hyperesophoria. In other words, the cobalt glass determines the fixing, and hence, also, the squinting eye. The reason for the association of hyperphoria with esophoria in hyperopia has already been explained, and an analogous explanation fits the association of hyperphoria with exophoria. If we admit that the tendency outward is a passive anomaly and due to a loss of convergence, this deficiency of innervation affects not only lateral but also vertical tensions, and where we should have in esophoria hyperphoria, we should expect to find in exophoria hypophoria.

Therefore, in considering the treatment of exophoria or esophoria, the condition of the vertical tensions must be thoroughly investigated and their causative relations determined where possible. The question often is raised, does the lateral imbalance depend upon the hyperphoria, or is the reverse true? The answer to this question depends upon the relative rotational powers of the vertical and lateral muscles. If the abductors and adductors show normal rotations (as determined by means of prisms and the tropometer) the elevators or depressors are likely at fault. If these latter exhibit normal and proportionate dynamic conditions, the lateral muscles are probably causative; for instance, Mr. L., aged thirty-one, a minister of the gospel and a close student, exhibits with the Maddox rod at 20 feet, exophoria of 1 degree and R. hyperphoria of 1 degree. At 20 inches the Maddox rod showed exophoria 12 degrees. Were now the lateral or vertical muscles at fault? Estimation of the prism power of rotation with rotary prisms showed: Abd. = 6 degrees; add. = 18 degrees; R. Supra = 3 degrees; R. Infra = 1 degree. The lateral muscles were therefore proportionate in their tensions and the difference of 2 degrees in the right supra- and infraduction was sufficient to fix the vertical muscles as the offending ones; in this particular case the fault consisted in either overaction of the elevators of the right eye or the depressors of the left eye—or underaction of the depres-

sors of the right eye or elevators of the left eye. This last point of overaction or underaction is to be determined by the tropometer.

Treatment.—The treatment of hyperphoria resolves itself into four phases.

1. Exclusion of any general disease or affection of the central nervous system, incipient tabes and general paresis in particular.

For instance: M. A., aged thirty-four, broker, referred by his family physician on account of eye symptoms, showed 4 degrees of left hyperphoria. No diplopia could be shown in any part of the binocular fixation field. There was no other abnormality about the eyes save incipient Argyll-Robertson pupils, which led to the suggestion that he consult a neurologist. One year later without the use of any correcting glasses (as he was practically emmetropic) the hyperphoria had disappeared entirely, but the Argyll-Robertson phenomenon was completely developed, and his accommodation was normal. Obviously it would have been bad practice to resort to either prisms or operation in such a case.

2. Thorough-going refraction just as in esophoria and exophoria, the correction being done under cycloplegia up to forty-five years of age. The persistent use of such corrections will suffice in many cases if not to partially or entirely dispel the hyperphoria, at least to render the patient so comfortable that treatment of the vertical imbalance as such need not be considered. We have already recited such a case history. It must be admitted, however, that correction of the refraction does not of itself so often prove the only necessary factor as it does in esophoria and exophoria. In our experience, esophorics profit most by a good correction alone, exophorics not so often, and hyperphorics least often.

3. If, after faithful use of a good correction and careful attention to life habits, the hyperphoria persists and is annoying, it is well to try the effect of a prism or prisms that correct about 1/3, of the hyperphoria. A convenient method is to have the prisms slipped into what is known as an extra-front, to be hooked over the regular glasses (Fig. 56). The prism effect may be divided between the two eyes (*base down* before one, *base up* before the other), or the whole prism to be used

may be placed *base down* before the hyperphoric eye, preferably the former for reasons to be stated later. By this means prompt and at times surprising relief is sometimes obtained, as, for instance, in the following case: F. E., aged twenty, a goldsmith, works all day at a distance of from 8 to 10 inches soldering small gold trinkets. Given eighteen months ago by a colleague (who used a cycloplegic) R. E. + .50 D + .50 Cyl 90 degrees L. E. + 0.75 D.S. With this correction he was much more comfortable at his work for two or three months, but his eyes and head began to trouble again, when his oculist, after much pains, ordered a 1/2 degree prism,

FIG. 56.—Hook or extra front.

base down, to be incorporated in the left glass, indicating that he found *left hyperphoria.* (This point is emphasized because of the subsequent developments in the case). Again he pursued his work with renewed comfort for about three months, when the old train of eye and head symptoms once more took up its march. At this time he came under our observation, and on September 14, 1897, he was carefully refracted under thorough cycloplegia and the findings of the previous worker were confirmed in all but the muscular details.

September 16, 1897. Maddox rod shows esophoria 1/2 degree. No hyperphoria. Abduction = 7 degrees. Adduction = 15 degrees. Left supra- and infraduction = 3 degrees.

September 30, 1897. Maddox rod used over patient's correction shows exophoria 1 degree. *Right* hyperphoria 1/2 degree. (*Left* hyperphoria had been found by his former adviser). With the parallax test R. hyperphoria 1 1/2 degrees. Exophoria 2 degrees.

October 14, 1897. Left hyperphoria 1 degree full, with parallax

test. Maddox rod shows vertical balance. Control tests made four times during the succeeding month showed R. hyperphoria 1 degree full with the parallax test, and each time R. supraduction equalled 4 1/2 degrees, while R. infraduction equalled 2 1/2 degrees.

This accord in results in four consecutive examinations justified having the 1/2 degree prism, base down, taken out of the correction of his left eye and having him try a 3/4 degree prism, base down, before the right eye in a slip front to be hooked on over his glasses while at work. One month later he stated that he had never been so comfortable. He was then directed to have the 3/4 degree prism, base down, incorporated into his right glass, because he was so much annoyed by the reflections from the prism in the extra front. Four months later the patient stated that he was working at his soldering bench (at a distance of 8 inches) for 10 hours a day, without discomfort of eyes or head.

This case illustrates how the most careful worker may be led into error by a spastic hyperphoria which we believe this young man to have shown in the earlier part of his trouble. Later, long-continued observation of his case and prism estimation of the upward and downward rotation showed not only right hyperphoria, but the difference between the right supraduction (4 1/2 degrees) and infraduction (2 1/2 degrees) pointed to the overacting elevators of the right eye as the cause of the deviation. Theoretically, tenotomy of the right superior rectus or of the corresponding inferior oblique was indicated, but prisms in the position of rest (favoring the underacting or weaker depressors of the right eye) gave the patient such prompt and lasting relief that operative measures were not to be thought of.

Naturally there are those who contend that the prism is but a crutch that must needs be added to from time to time to the great disadvantage of the patient's ocular muscles, but as there is almost no other alternative but operation, prisms must be considered and must be offered to the patient as a possible means of relief. Also there are those who claim that if in a given case 2 degrees of hyperphoria are definitely shown, that a 2 degree prism should be

incorporated in the patient's lenses to establish vertical balance once and for all. From the academic standpoint this would seem to be perfectly reasonable, but abundant experience has shown that the average patient glides much more easily into the comfortable use of vertical prisms if one-third to one-half of the deviation for distance is employed, having the patient to understand that as time goes on more prism strength may be demanded by the eyes.

Latent hyperphoria is ofttimes uncovered by the latter process and there is every reason to believe that once a patient's full hyperphoria has been learned by this method there is little likelihood that any more deviation will reveal itself no matter how long the case is studied. We have a long series of cases in support of this belief. Yet warmly as we wish to endorse the practice of temporary trial of prisms in the hook front, there are many individuals whose symptoms are only aggravated by this measure, in which case exercise of the vertical muscles may be tried by means of prisms placed base up or down, as the case requires. Savage believes this method to be of value in deviation tendencies under 1 1/2 degrees, and says: "The prisms used should range from 1/4 to 2 degrees; most cases will not require stronger than a 1 degree prism. The apex of the prism should be placed in the direction of action of the muscles to be developed, the patient exercising from two to ten minutes, two to five times daily. The object looked at must be 20 feet distant, and it should be seen *through* the prism 5 seconds, and then *without* the prism 5 seconds, and so on throughout the sitting." While our own experience with this method in hyperphoria has been limited, our results have not been such as to lead us to expect much relief from this kind of training.

If, then, a patient presents persistent hyperphoria and will tolerate neither prisms in position of rest nor exercise of the weaker muscles, are we to turn immediately to operation as a last resort? Not so. Many factors must be determined before operative treatment is applicable. For instance, it is not to be thought of when the hyperphoria is recent, progressive, or variable; in case of

central nervous disease, in rheumatic, gouty or diabetic subjects, or in those who have not enjoyed binocular vision for years (strictly speaking, these are instances of hyper*tropia*). Moreover, when hyperphoria complicates esophoria or exophoria, it is not infrequent for the vertical anomalies to right themselves, or at any rate for the symptoms attributed to them to disappear when the lateral muscles have been well trained with prism gymnastics. Particularly is this true when exophoria coexists.

4. *Operation.*—Hence we find operative treatment of the hyperphorias narrowed down to the permanent, constant or static variety, where the deviation tendency is constant in amount and character notwithstanding at least six months of the proper correction, where all tests agree in showing marked over or underaction of one set of elevators or depressors, when prisms have not been well borne, and exercises are of no avail.

In the former set of cases (overaction) tenotomy is highly satisfactory and often brilliant in results, if the case has been selected in accordance with the above suggestions. The dissipation of reflex symptoms following upon tenotomy for hyperphoria is sometimes little short of wonderful, and has doubtless led many witnesses of such cures into operative treatment of hyperphorias that are distinctly outside of the operative class, and given the extreme illogical conservatives in ophthalmology occasion to deride the judicial and unjudicial alike as "muscle-snippers."

Hyperphoria, constant in degree and kind, definitely due to underaction on the part of the vertical muscles, may be met by advancement or muscle shortening of the underacting muscle or muscles, and in properly selected cases the same results may, as a rule, be expected. It should be borne in mind, however, that advancement of a vertical muscle is not so easily performed as on a lateral one and this may influence the surgeon to resort to tenotomy of the opposing muscle.

Operation, whether tenotomy or advancement, should always aim to slightly under-correct the deviation, the remainder being often amenable to prism exercise of the weak muscle immediately

and from twenty-four to forty-eight hours after the operation; in this way the effect of an operation that is insufficient may be considerably increased; any slight remaining deviation can be readily met by the necessary prism in the position of rest.

Surgeons of large operative experience with muscular anomalies prefer to aim at a slight over-correction of the imbalance, because, as they assert, reattachment of the operated muscle is almost sure to be attended with some loss of the original effect. The practice is a safe one, however, only in the hands of those who have perfected themselves in the technique of operations on the ocular muscles.

DECENTERING LENSES.

Some oculists prefer to decenter spherocylindrical lenses rather than to write for the lens and the prism separately. Especially in Great Britain is this in vogue. If this practice is to be followed it is well to bear in mind the general rule that for every centimeter (or 10 mm.) of decentering there will result as many prism degrees as there are diopters in correcting lens. Thus:

+4.00 sphere $\bigcirc$ 4 degree prism base out may also be written
+4.00 sphere decentered 1 cm. outward, (or 10 mm.).
or +4.00 sphere $\bigcirc$ 2 degree prism base out may also be written
+4.00 sphere decentered 5 mm. outward.

While the ophthalmologist may occasionally have recourse to this method of securing a prismatic effect we do not recommend it as a routine measure. Opticians in this country are in the habit of receiving prescriptions as follows:

+2.00 sphere + $\bigcirc$ 0.50 cyl. axis 45 degrees $\bigcirc$ 1 degree prism *base in.*

and they then decenter the lens to produce the proper effect. The table prepared by Dr. Edward Jackson here shown indicates the amount or decentering of a lens of known focal length to produce a given prismatic effect.

Jackson: Decentering of Lenses for Prismatic Effects, with Glass having an Index of Refraction of about 1.54.

Power of lens in diopters	To obtain 1° prism	To obtain 2° prism	To obtain 3° prism	To obtain 4° prism	To obtain 5° prism	To obtain 6° prism	To obtain 8° prism	To obtain 10° prism
	Decenter mm.	Decenter mm.	Decenter mm.	Decenter mm.	Decenter mm.	Decenter mm.	Decenter mm.	Decenter mm.
1 D.,	9.4	18.8	28.3	37.7	47.2	56.5	75.8	95.2
2	4.7	9.4	14.1	18.8	23.6	28.2	37.9	47.6
3	3.1	6.3	9.4	12.6	15.7	18.8	25.3	31.7
4	2.3	4.7	7.1	9.4	11.8	14.1	18.9	23.8
5	1.9	3.8	5.7	7.5	9.4	11.3	15.2	19.
6	1.6	3.1	4.7	6.3	7.9	9.4	12.6	15.9
7	1.3	2.7	4.	5.4	6.7	8.1	10.8	13.5
8	1.2	2.3	3.5	4.7	5.9	7.1	9.5	11.9
9	1.	2.1	3.1	4.2	5.2	6.3	8.4	10.5
10	.9	1.9	2.8	3.8	4.7	5.6	7.6	9.5
11	.9	1.7	2.6	3.5	4.3	5.1	6.9	8.7
12	.8	1.6	2.4	3.1	3.9	4.7	6.3	7.9
13	.7	1.4	2.2	2.9	3.6	4.3	5.8	7.3
14	.7	1.3	2.	2.7	3.4	4.	5.4	6.8
15	.6	1.3	1.9	2.5	3.1	3.8	5.1	6.3
16	.6	1.2	1.8	2.4	3.	3.5	4.7	6.
17	.6	1.1	1.7	2.2	2.1	3.4	4.5	5.6
18	.5	1.	1.6	2.1	2.6	3.1	4.2	5.3
19	.5	1.	1.5	2.	2.5	3.	4.	5.
20	.5	.9	1.4	1.9	2.4	2.8	3.8	4.8

On Testing Prismatic Lenses.

The prescriber should be prepared to test the correctness of prismatic lenses when they are returned to him for inspection.

A common usage is to select some object in the room with a long straight side to it such as the door jamb. With the spherocylindrical element of the lens properly neutralized the optical center of the whole combination is held before the observer's right eye and in line with the door jamb, when that portion of the

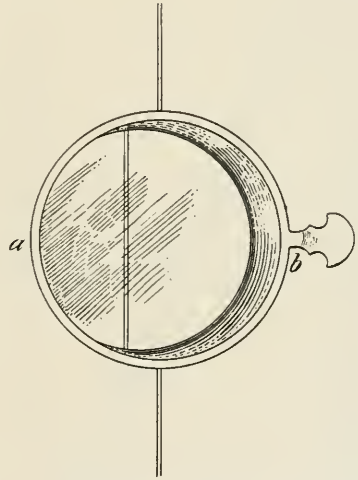


FIG. 57.—Effect of a prism in breaking the edge of a door jamb or any straight line.

edge of the jamb seen through the lens will appear displaced to one side. (Fig. 57). The prism that will restore the displaced portion of the door jamb so that it appears continuous will be the measure of the prism that is incorporated in the lens.

This same principle has been utilized by Zeigler in designing his card for measuring the prism strength of such lenses. After the spherocylindrical portion of the lens has been properly neutralized as in the preceding experiment, it is held at one or two meters from the card (depending on which distance the card is

adapted for) and the prism strength immediately read off according to the amount of displacement produced. It is convenient, accurate and serviceable (Fig. 58).

One other method remains, the use of the douziememeter.[1] The instrument is applied to first the nasal and then the temporal edge of the lens, both of which should measure the same

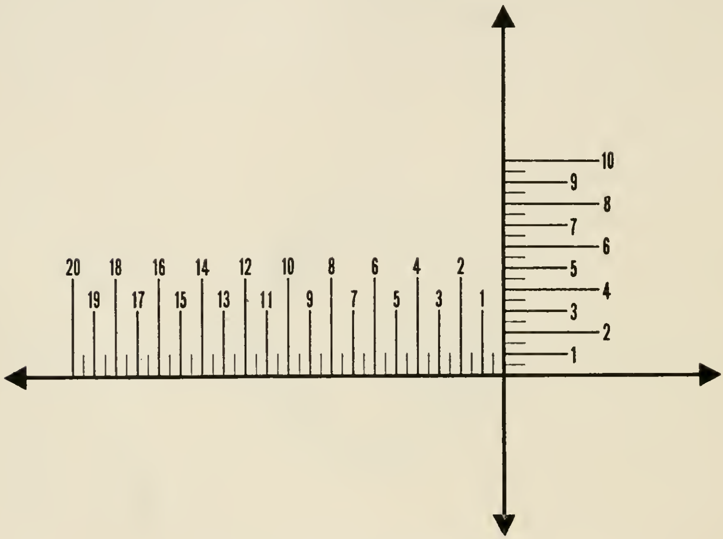


Fig. 58.—Ziegler's prism measure card.

in case there is no prism element in the glass (Fig. 59). The same is true of the upper and lower edges of the lens. If the douziemeter registers say 10 douziemes for the nasal edge of a lens, and 6 douziemes for the temporal edge, the nasal edge being 4 douziemes thicker represents a 1 degree prism base in. A lens that is 2 douziemes thicker at the nasal than at the temporal edge would represent a 1/2 degree prism base in and a lens 8

[1] The name is derived from the French douzieme—or one-twelfth—having reference to the old system of twelve lines to the inch. The scale of the douziemeter is marked in twelfths of an inch, or lines.

douziemes thicker at the nasal than at the temporal edge would represent a 2 degree prism base in. The same method of measurement holds in measuring the upper and lower edges to detect any vertical prism that may be present in a lens.

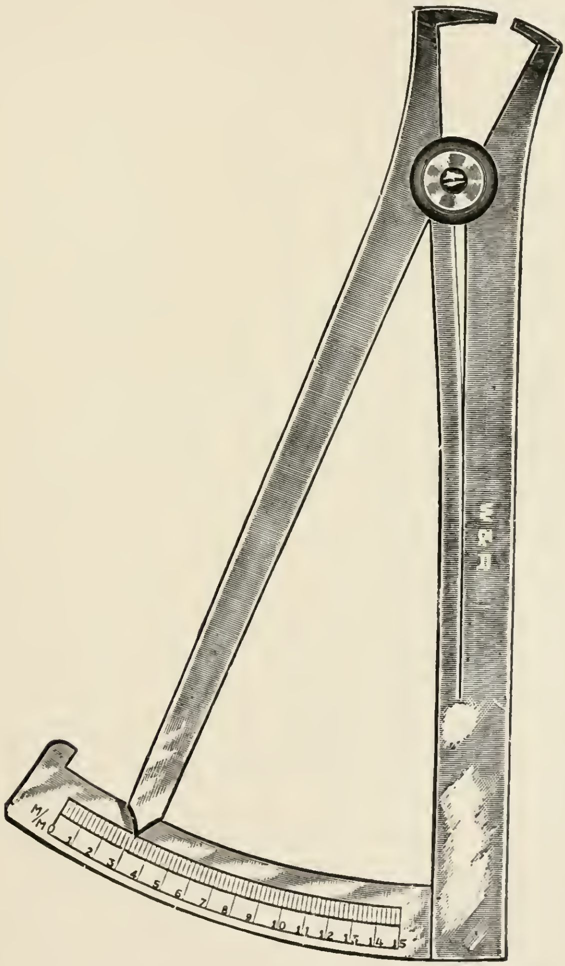


FIG. 59.—The douziemeter.

HETEROTROPIA.

ESOTROPIA.[1]

Esotropia manifests itself externally by an inward deviation of one cornea, or the inclination of the visual axes toward each other, so that they cross at some point inside of infinity; or more correctly speaking, the visual axis of one eye is directed toward that of the other.[2]

Etiology.—The factors that individually or in combination produce esotropia are:

(a) Refractive errors (generally H and Ah conditions).

(b) A congenitally weak fusion-faculty.

(c) Congenital paresis of one or both external recti (to be distinguished from true congenital abducens palsy).

(d) Obstetric injuries to the eyes.

Refractive Errors.—The usual refractive status in most subjects of esotropia is that of compound hypermetropic astigmatism. The error need not necessarily be high. Many esotropic children do not manifest more than 2 diopters of error. In the cases that are solely due to the refractive error the correction of the abnormal refractive status with suitable glasses generally causes the strabismus to rapidly disappear. This variety conforms absolutely to Donder's theory as to the dependence of the deviation on the abnormal convergence stimulus set up by the refractive error, and may properly be called accommodative esotropia. Not all cases of esotropia, however, are explainable on this basis. Ninety-six per cent. of all children are born hypermetropic and the question may justifiably be put, "Why do not all hypermetropic children show esotropia?" Some other factor must, therefore, be operative and the most probable one is a *Congenitally Weak Fusion Faculty.*

[1] Synonyms.—Internal or convergent strabismus; internal squint.

[2] The student is urged to read the chapters on "The Evolution of Binocular Vision" and "The Relation between Accommodation and Convergence" before entering upon the study of esotropia.

There seems to be much basis for the supposition that certain children exhibit a very weak fusion faculty.

Whether this is due to hereditary influence or to imperfect development of the entire visual apparatus during fœtal life is still a mooted question. Nevertheless, the fact remains that a goodly percentage of esotropic children are the subjects of this weakness, in consequence of which the child's eyes do not acquire normal conditions and sooner or later begin to exhibit abnormal deviation (generally in the direction of convergence). It will be readily seen, therefore, that in this variety of cases it matters little whether the refractive condition is hypermetropic, emmetropic, or myopic, the deviation develops quite the same; as for instance in the following case:

H. G., aged three, brought because of an esotropia that had begun to appear about a year previously. The little patient was a fine, lusty, healthy boy, whose personal and family history were good and whose eyes seemed to be normal in every respect save for the deviation, which equalled 40 degrees. Under thorough atropinization the eye-grounds were found normal, and the retinoscope showed only one-half a diopter of hypermetropia without any astigmatism. In the presence of so insignificant a refractive error some other factor had to be sought for and it was found that his fusion faculty was practically absent. Obviously the treatment had to be carried out without the help of any correcting glasses.

Or esotropia may be found in a child with myopia, as follows:

W. S., aged four, a strong, healthy child began to show occasional deviation of either eye near the end of his second year. There was no illness, no trauma; in fact, no contributing factor of any kind that could be made out. He had three sisters, all of whom presented normal eyes. His deviation amounted to 30 degrees in spite of the fact that the temporal rotation of each eye seemed normal. Under thorough cycloplegia the eye-grounds were found normal and the retinoscope established his refractive status as

R. – 1.00 sph. – 75 cyl.axis 45 degrees
L. – 0.75 sph.—50 cyl.axis 180 degrees.

With his correcting glasses 10 degrees of the deviation disappeared, leaving 20 degrees in evidence and it so remained at the end of a year. Since that time some education of the fusion faculty has been accomplished and today (after five years) the deviation equals 10 to 12 degrees. He now has a fair chance to reach manhood with an approximately straight pair of eyes and without having been subjected to operation.

Necessarily such cases are rare. The usual condition is that of a somewhat subnormal fusion faculty that is embarrassed in its evolution by an unequal compound hypermetropic astigmatism. Indeed, it is our feeling that this is the usual complex in the vast majority of cases presenting themselves either in private or clinical practice.

Congenital Paresis of One or Both External Recti.—As stated in the classification, this is to be distinguished from true congenital abducens palsy. It is not frequently encountered, but one should nevertheless be on his guard. For instance:

Miss H. C., aged twenty-six, comes for advice as to her esotropia, which had shown itself mainly from her fourth year onward. At her sixth year she was refracted and for twenty years she had worn glasses that so lessened the esotropia as to entirely satisfy her as to her appearance. She showed 15 to 20 degrees of esotropia, varying from time to time. Her refractive status was typically H and Ah, for which she was wearing a good correction. She preferred to fix with her right eye, although the vision was normal in both eyes. With the tropometer it was found that her temporal rotation in either eye was but 20 degrees (as against the normal 50). Homonymous diplopia was found in the extreme temporal periphery of both motor fields, although it was not noted until the light was 30 to 35 degrees from the median line. It would have been an easy matter to overlook the somewhat defective temporal rotations in these eyes. Muscular advancement of both external recti in this case entirely corrected the defect.

Obstetric Injuries to the Eyes.—Within recent years Wolff and von Sicherer have studied the eye-grounds of newborn children

and have found numerous instances of retinal hemorrhages not only in children who were instrumentally delivered, but also in those who were born without the use of instruments. These facts may have much bearing on the genesis of esotropia. If a child should be born with an extensive retinal hemorrhage involving the macular region and this hemorrhage were imperfectly absorbed it is not difficult to conceive that there might be sufficient hindrance to the normal evolution of binocular vision to induce suppression of the images from that eye such as occurs in children with a defective fusion faculty, in which event the

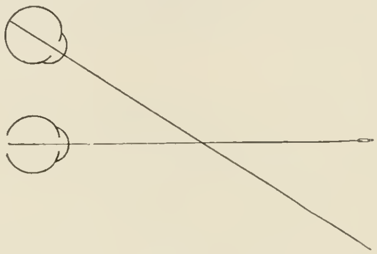


FIG. 60.—Crossing of visual axes.

eye so affected might eventually deviate from parallelism. If the child had a congenitally strong fusion faculty the eyes would be held parallel, but the child would grow up with a congenital amblyopia. It is probable that this is the explanation of that class of cases that come to report from time to time, of amblyopic eyes seemingly without any ophthalmoscopic change and yet revealing a well marked central scotoma.

It must be borne in mind always that the image of the object engaging the attention of the patient falls upon the fovea of the fixing eye, and upon some portion of the retina to the nasal side of the fovea of the squinting or deviating eye. Hence, the visual axes are not, strictly speaking, inclined toward each other; but the axis of the fixing eye is directed straight at the object, while that of the deviating eye intersects that of the fixing eye at a point nearer the face than the object; in other words, both eyes are not abnormally converged at the same moment (see Fig. 60).

The burden of the convergence is borne by one eye only, transferable under some conditions to the other, but never manifested simultaneously in both. This statement is true only of the properly so-called functional esotropias, and excludes paralysis of the external muscles and association paralysis.

Esotropia is *monocular* (or constant), and *binocular* (or alternating). In the former, one and always the same eye, under all circumstances is used for fixation, while the former eye deviates. In the latter, either eye is used indifferently for fixing, and the squint is instantaneously transferred to the eye not so used.

Monocular Esotropia.

Monocular esotropia is characterized by (1) lowered vision or even amblyopia of the squinting eye; and (2) invariable fixation of the other eye.

Diagnosis.—The methods that take into consideration the apparent deviations of the visual axes are at times misleading. For example, the angle alpha made by the intersection of the optic with the visual axis may be abnormally small, giving the appearance of internal strabismus, while the muscles may be in equilibrium. Too, the interpupillary distance, the shape of the orbit, the size of the commissure and other anatomic peculiarities should always be taken into consideration.

Fig. 61.—Reber's object or kindergarten test card.

The three facts that should be determined in every case of esotropia are the vision, the degree of deviation, and the refractive status.

Vision.—In children under three years of age, estimate of the vision is most unsatisfactory and generally impossible. Between the third and the seventh year, some form of object test card will be necessary. Fig. 61. The one illustrated we have found of great service. Naturally much tact is required in handling these little people, but judgment and patience will be amply repaid when the coöperation of the child is secured; this once accomplished all the rest is easy. In esotropic children under six years of age, the vision in the

deviating eye will seldom be found less than 5/30. After the seventh year and on up to puberty it frequently falls to 5/60 and at times to 2/60 or 3/60.

Amblyopia.—No term in all ophthalmic literature has been more loosely used than this term amblyopia. While it admittedly means "indifferent vision," few writers seem to have stated just what degree of loss of vision shall justify our use of the term amblyopia. Inasmuch as vision of at least 1/4 (5/20 or 20/80) is essential to permit comfortable use of an eye for reading, it would seem justifiable to fix that as the arbitrary mark. Vision of less than 5/20 or 20/80 could be definitely set down as amblyopia and more than that as not amblyopic. It is understood that the ophthalmoscope shows no opacities in the media, and that the details of the eye-ground conform in every particular to the normal standard, although the field of vision, as above stated, is sometimes defective, especially in its central portion.

That no minute anatomic changes of the optic nerve occur in the fovea or in the foveal fibers seems clear from the report of several cases in which an amblyopic squinting eye was restored to useful vision in the course of a few weeks by its enforced use. Again, the error of refraction of the amblyopic eye not infrequently differs very little from that of the seeing eye, certainly not enough to account for that grade of defect which will exclude it from all participation in vision. It is probable that in consequence of a functional (but not anatomic) defect in the basal ganglia or the cortical centers for vision, acting in conjunction with a degree of hypermetropia high enough to disrupt the association of convergence and accommodation, one eye has been excluded from vision and its function held in abeyance. Whatever the explanation may be, it is true that amblyopia is invariably associated with monocular squint, that it limits the squint to one eye, and stands in the way of a scientific correction of the muscular error, since binocular vision is practically unattainable.

In some cases restoration to parallelism of the visual axes can, in fact, be effected, only for a brief period however, since the blurred image seen by the amblyopic eye interferes with the clear

one seen by the good eye, and in the interest of good vision the indistinct image comes to be disregarded and an internal or external deviation is developed. Improvement has been reported as the result of exclusion of the good eye, enforcing education and training by constant use of the other; it has also been claimed as a result of operation, but in our experience the means often successfully employed to correct squint in childhood without operation, such as atropin, bandaging of the good eye, etc., have proved indifferent in curing amblyopia, and the hope that vision may be restored to a practical and useful extent by these means has not been entirely fulfilled. Amblyopia does not signify blindness (for which the obsolete word is amaurosis), and when not qualified by an adjective, such as toxic, renal, etc., indicates simply that the patient's vision is defective from no known causes. The use of the word should be limited to those eyes whose vision is lower than 20/80. Perseverance scientifically applied will often be rewarded by a decided improvement of vision by correcting lenses.[1]

Measurement of the Deviation.

The means employed for the measurement of the deviation are: a. inspection, b. the cover test, c. linear measure, d. the perimeter or arc measurement, e. the tape measurement or tangent measurement, f. the diplopia test, g. the tropometer or rotational test.

a. *Inspection.*—Inspection will show a want of coördination of the visual axes by the fact that the sclera of the temporal side of the deviating eye is exposed in greater extent than that on the nasal side; furthermore, that the cornea is deflected toward the nose.

b. *Cover Test.*—The patient fixes the gaze on some object 20 or more feet distant. The cover or screen is then placed over the fixing eye compelling the previously deviating eye to fix, when it will be seen to move from 2 to 5 mm. to reach the proper position

[1] Should the reader desire to pursue the theories of amblyopia farther than they are here discussed, he is referred to the papers of Hansen Grut, Schweigger, Schmeichler, Priestley Smith, Parinaud, Javal, Worth and others.

for fixation. The degree of deviation can be estimated with fair accuracy by interposing prisms with their bases out, increasing in strength until all movement of either eye (as the screen is carried to and fro in front of the eyes) is abolished. The prism strength required to stop the movement of either eye is the measurement of the deviation.

c. *Linear Measure.*—Years ago von Græfe devised a small curved ivory scale marked in millimeters to be laid against the lower lid and the deviation then read off in millimeters (Fig. 62), but, as Landolt insists, it is not exactly scientific to speak of an angular

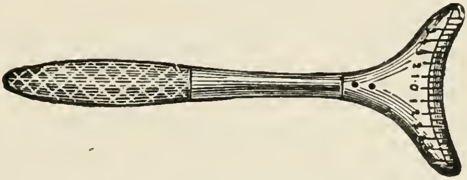


FIG. 62.—Graefe's strabismometer.

deviation in linear terms. This may be obviated by utilizing the images formed by the corneas when a strong light is thrown on them. With the light directly over the patient's head, a retinoscopic mirror will produce quite a bright corneal image if thrown upon the eyes from one meter's distance. Hirschberg estimated that this reflection image, if seen at the corneal limbus represented a deviation of about 45 degrees; if on the sclera it is 60 to 80 degrees; if midway between the limbus and the margin of an average sized pupil it is about 15 to 20 degrees, and if at the outer edge of the pupil about 10 degrees. With practice a fairly accurate estimate may be made.

d. *The Perimeter or Arc Measurement.*—The patient is seated at the perimeter just as for ordinary perimetry only that both eyes are kept open. The non-squinting eye is then fixed upon some object at infinity directly in line with the center of the perimeter arc while the observer moves a small electric light along the arc of the perimeter. The image formed by this light on the cornea of the deviating eye is studied and the light moved backward and

forward along the arm of the perimeter (which should be in the horizontal meridian) until the tiny corneal image rests directly in the center of the pupil of the deviating eye, when the observer notes at what degree on the perimeter arc the light is resting. This is the true measure of the deviation in degrees. (See Fig. 63). The perimeter method is easily applied in grown children and adults, but is of little use in children under six years of age.

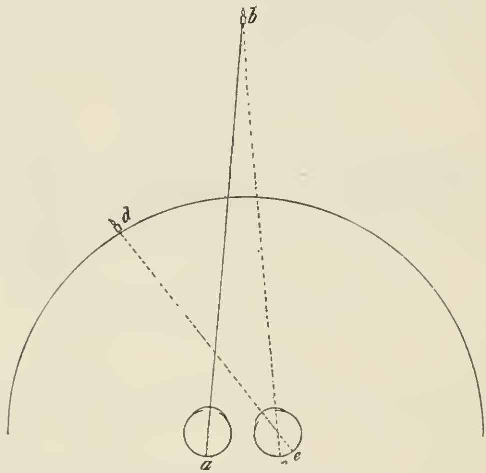


FIG. 63.—Arc measurement of the deviation in esotropia. The arc of the perimeter is in the horizontal meridian.

e. *Priestley Smith's Tape Measure Method* (tangent measurement).—In a darkened room the observer takes a position one meter from the seated patient, over and back of whose head is a light. The observer is armed with a retinoscopic mirror and a tape measure marked in centimeters. The patient gazes at an object at infinity. The light is thrown by the mirror on the deviating eye when the corneal image will be seen near the limbus of that eye. With the tape measure held at zero against the handle of the retinoscopic mirror, the observer moves his disengaged hand horizontally away from the direction of the deviating eye, directing the patient to follow his moving hand (through which the tape measure is allowed to slide) until the deviating eye is

brought into such a straight ahead position that the corneal image rests just in the center of the pupil of that eye. Then the observer's moving hand pinches the tape measure wherever it may be at this instant. The number of centimeters the moving hand has covered on the tape measure records the number of degrees of deviation present. This method is quick, accurate, convenient and quite as applicable in a three year old child as in an adult.

f. *The Diplopia Test.*—By determining the relation of the image of the squinting eye to that of the fixing eye, the diagnosis of the actual position of the squinting eye and the degree of variance of its visual axis from parallelism can often be made. In amblyopics, the surgeon experiences the greatest difficulty in forcing the patient's recognition of the image of the amblyopic eye because, as has already been stated, he never complains of diplopia, nor is he ever conscious of the false image of any object. Therefore, examination by means of double images would seem to be impracticable, but in our experience persistent efforts have frequently accomplished the end sought. At first, the patient absolutely refuses to acknowledge the false image, but with the aid of strong prisms, *base out*, before the squinting eye (bringing the false image nearer to the true) and by means of colored glasses (particularly those that render the true light dull and indistinct) perseverance will finally be rewarded with the acknowledgment that the object has its shadow. Having once secured this recognition, the subsequent steps present no difficulty. The prisms, bases out (and bases down if necessary), before the squinting eye which will fuse the images will be the prismatic measure of the deviation. By pursuing this course another important factor in the diagnosis, viz., the difference of elevation of the true and false light, is readily and accurately determined.[1] The false image will be on the side of the squinting eye (homonymous) and often below that of the true eye. The lateral deviation equals 20 degrees to 40 degrees or more, and the vertical difference is usually overcome by a prism of 3 to 4 degrees. This determina-

[1] Cases that prove exceptions to this rule are those of false projection (the so-called second fovea), and those of mental incapacity.

tion of hypertropia is necessary to the treatment, even though the result of the operation can only be cosmetic. Clinically, we have found that vertical squint is not transferred with the lateral in cases of high amblyopia, the eyes retaining the same relative position even when the sound eye is covered.

g. *The Tropometer.*—While this is a supplementary test the information furnished by this instrument is of value in indicating the exact power of temporal rotation of esotropic eyes. Its findings generally show subnormal power of temporal rotation in such cases. The motor fields here shown are from a typical case of monocular esotropia (Fig. 64).

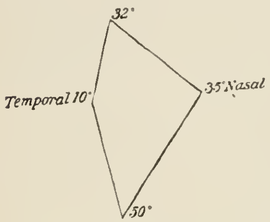


FIG. 64.—Tropometric fields in a case of monocular esotropia (right eye deviating eye). The average normal rotations are upward 36, downward 52, temporalward 48, nasalward 50.

Another supplementary test is the deviometer of Worth (modified by Black), but it serves no purpose not fulfilled by the foregoing tests and simply multiplies office apparatus.

Treatment.—The management of monocular esotropia is nonoperative in the developmental and operative in the confirmed stages.

The nonoperative treatment is applied with the hope of accomplishing two purposes, namely: the subsidence of the squint and the cure of the amblyopia. Early in the history of the squint the peripheral cause, hyperopia, can be set aside by paralyzing the accommodation, thereby silencing the abnormal stimulus to convergence always consequent upon the unconscious but unceasing overaction of the ciliary muscle. It is not uncommon to see an esotropia disappear in small children after two to three months paralysis of the accommodation and use of a proper correction. The drug usually employed for this purpose, and the one best borne by children without serious results after prolonged application, is atropin, which it must be said is not without its disadvantages. The pupils partake in the paralysis and become widely dilated, giving rise to photophobia; the follicular glands of the conjunctiva take on a hypertrophic inflammation, and the

squint, for a time at least, seems to be increased because, although the impulse to accommodation is not interrupted, accommodation itself (response to the impulse) is impossible. On the other hand, the associated impulse to convergence is greater because of the patient's efforts to correct his defective vision by calling urgently on his ciliary muscles. However, this effect of atropin is transient and may with safety be disregarded. It may be applied either to both eyes or only to the fixing eye. In the latter case inducement is given the patient to use the squinting eye, provided that its vision is better than that of the fixing eye when atropinized. For the same reason periodic wearing of a bandage or screen over the sound eye is advocated to promote the retinal functions of the deviating eye. It is our opinion that in monocular squint with its attendant amblyopia, this preliminary treatment while not often effectual in curing the deformity or in restoring binocular vision, is certainly useful in preventing an increase in the ametropia and a decrease in the vision of the amblyopic eye. The therapy of esotropia further includes the optical correction of the refraction error, even in very young subjects.[1]

This should be invariably determined by the ophthalmometer and retinoscope and in children under six years of age it is the part of wisdom to bind off one eye while retinoscoping the other. No satisfactory treatment of esotropia in children can ever be instituted without the use of the retinoscope under thorough cycloplegia. A full correction, or as nearly full as possible, should be worn *constantly*. The beneficial effect of glasses may be increased by the joint use of atropin. And yet, however essential these means may be in the early treatment of monocular esotropia, they are seldom final, either singly or jointly. As long as the vision of the fixing eye is more acute than that of the squinting eye, the patient will prefer to use the better organ, and neither the squint nor the amblyopia, will be cured, although much progress in this direction will often be secured by employment of the stereoscope, as advocated by Landolt or the amblyoscope as used

[1] Gould has recently (*Phila. Med. Jour.*, May 18, 1898) reported cases in which glasses were successfully worn for squint as early as the twenty-ninth month of life.

by Worth (see Fig. 65) for education of the fusion faculty. The latest model of the Worth amblyoscope is arranged with a double rheostat so that the intensity of illumination in either tube can be perfectly controlled by simply pushing a steel collar to and fro. In this way the intensity of retinal stimulation may be beautifully equalized. No other device at present offers this advantage. Three sittings a week for four weeks should be required of the patient. If at the end of this time no improvement is noted surgical interference must be considered. At this point several questions are suggested, viz.: At what age is operation to be recommended? To what muscles should the treatment be directed? Should it include one or both eyes?

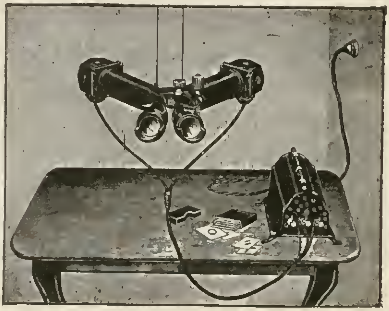


FIG. 65.—Worth's amblyoscope with rheostat.

Authorities state that six years is the earliest age at which operation ought to be performed, and that nine to ten is the most advantageous. The advocates of early operation urge that a restoration to parallelism of the visual axes will prevent the development of amblyopia, which they claim ensues from non-use. The objections to early operation are: the immaturity of the globes, the natural increase in the power of the muscles of growing children, the impracticability of accurately estimating the degree of the squint, and hence the effect desired or secured by operation; also the fact that when tenotomy of the internal recti is performed crudely, too extensively, or at too early an age, divergence often ensues between the twentieth and thirtieth year. In esotropia,

as in esophoria, the surgeon has the choice of tenotomy of the interni, or advancement of the externi, or a combination of both. Inasmuch as a scientific restoration of binocular vision cannot often be obtained in monocular squint, and as all procedures are carried out with the object of removing the deformity, it has been argued that attention should be directed mainly to the squinting eye. But tenotomy of the internus of this eye only will be productive of little good, and it will nearly always be necessary to tenotomize the internus of the other eye also. While in many instances the visual axes may be brought to parallelism by these two operations, it will often be accomplished only by such thorough division and separation of the interni from the neighboring tissues, that the effect is practically that of paralysis. In a few years the evil consequences of such measures crop out either in divergence of the amblyopic eye, or serious impairment of the inward rotation of both globes. The proceeding that appears to offer the greatest advantage, both immediate and permanent, is advancement of the externus of each eye, combined with a moderate tenotomy of the interni of the squinting eye. Subsequently, if necessary, tenotomy of the internus of the fixing eye may be performed. If vertical deviation co-exist, as it nearly always does, it is good practice to divide the superior rectus of the squinting eye first, since it has been shown in many cases that when hypertropia has been eliminated esotropia diminishes, and sometimes even disappears.

Alternating Esotropia.

Etiology.—Alternating esotropia differs somewhat from the monocular form in its clinical aspects and its treatment. For instance, disease of the foveal fibers of the retina and optic nerve of one eye (the result of congenital causes, of accidents during delivery, possibly of non-use of the eyes) by some authorities given much prominence in the development of the monocular, play very little part in the alternating form; in the latter variety, therefore, amblyopia (in its strict sense) is not to be expected. Moreover, in alternating esotropia the deviation is perfectly and readily transferred from one to the other eye. Finally, the results of treatment, both in permanently correcting the deviation and in re-establishing binocular vision, are infinitely more favorable in this variety. As to etiology, it may be said that occasionally an inherited deficiency or coördination in the cortical centers of fusion is undoubtedly a factor contributing to the loss of binocular fixation, although the usual exciting cause is a hyperopia of from 2.00 to 4.00 D. The former is plainly shown in the following case: E. F. M., a seven-year-old girl is brought with a history of esotropia appearing during the second year of life. There was nothing in the history that would account for the deformity. The child would fix indifferently with either eye. The vision equalled 5/5 in each eye, the deviation measure 35 degrees, with both eye-grounds normal in every respect. Temporal rotation in each eye was somewhat defective. Under thorough cycloplegia the refractive status was found to be + 0.50 sphere in each eye. Plainly the refractive error was not an etiologic factor. Investigation showed that the child's fusion

[1] *Synonyms.*—Concomitant internal squint or strabismus, alternating internal squint or strabismus, and insufficiency of the externi. (It is to be hoped that the expressions "concomitant" and "strabismus" as applied to esotropia, will soon be discarded.

faculty was practically nil. Fusion training was tried for eight weeks, but without avail and operation became the only resort. Advancement of each externus gave a most satisfactory result.

Exceptionally, the degree of hyperopia is much higher than 4.00 D., but esotropia is uncommon in very high hyperopia, unless the latter is attended with congenital disease of both foveæ (Reber). As in esophoria and monocular esotropia, the overaction of the ciliary muscle necessary to acute vision stimulates the nucleoli of the 3rd nerve, and through them the muscles governed by the nerve, with a resultant inward and upward deviation of either eye indifferently; therefore, the squint is transferable. (Many of these cases conform absolutely to the requirements of Donder's theory; that is, they are accommodative esotropias).

Diagnosis.—The methods employed in the recognition of monocular are equally applicable to binocular esotropia, and their detailed description may, therefore, be omitted.

As has been said above, fixation is quite indifferent, and the slightest circumstance may suffice to so disturb it that the squint is taken up by the eye which, up to that moment, had been fixing. It cannot be too strongly emphasized that the upward squint is also transferred, as can be readily demonstrated by testing with glasses of different colors. For instance, with a red glass before the right eye and a blue glass before the left eye, recognition of double images will be almost immediate. If the right be the fixing eye, the blue light (L. eye) will be to the left side of and below the red light. However, if the patient's attention is now directed to the blue light, making it the fixing eye, the red light (R. eye) will be to the right of and below the blue one, clearly showing that each eye deviated not only in, but also up, when the other was fixing. The lateral deviation varies from 20 degrees to 30 degrees and sometimes even to 50 degrees, while the vertical deviation is usually corrected by a prism of from 3 to 4 degrees. In conducting this test, the surgeon will sometimes have difficulty in bringing the patient to recognize the two images, but if diplopia can be induced no method of diagnosis is more accurate. In children of suitable

age for operation the false light will occasionally be suppressed at first, but after some little effort it will be recognized.

It will thus be seen that a description of esotropia naturally includes a description of hyperesotropia, and that esotropia as a pure lateral deviation rarely exists as a functional anomaly. The upward deviation has been noticed by few and overlooked by many writers, for the reason that the inward turn gives rise to a deformity so great that it masks the comparatively insignificant upward turning.

Treatment.—As has been said in the treatment of monocular esotropia it is not uncommon to perfectly and permanently cure this deformity in small children even when alternating, by paralyzing the accommodation for some months early in the history of the squint. It is scarcely necessary to add that the patient should wear, from the earliest possible moment, an accurate correction of his hyperopia or hyperopic astigmatism. The act of binocular vision is favored by the normal bilateral use of the accommodation which the glasses re-establish and by the increased visual acuity which they confer. These should never be omitted, even in those in whom the deviation seems to be fixed. Failing in these means, resort must be had to

Operation.—It may be well to repeat that squint is not primarily a defective muscular condition, and even less is it an affection that can be attributed to any one muscle. It is a matter of convergence power as related to divergence power, or still more accurately it is to be referred to the muscular response of excessive or deficient nerve stimulation. Therefore, it would seem, reasoning a priori, that the remedy is not to be found in operation on either the interni or the externi, but in regulating or coordinating the stimulus to convergence and divergence so that these functions may respond relatively equally and hold the visual lines in horizontal equilibrium. This is perfectly true and is carried out in practice, removing—by means of cycloplegics, convex lenses, muscular and fusion exercises and the efforts to improve vision—the causes of esotropia. But these measures often fail and operative measures only remain. In our judgment

we have not the choice between tenotomy or advancement when the convergence exceeds divergence by 20 degrees or more, which is always the case in functional esotropia. To cut the interni at their insertion so that an effect of 20 degrees or more is obtained

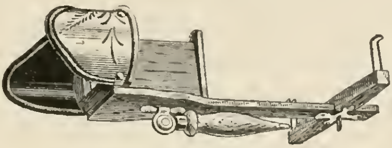


FIG. 66.—Holmes' stereoscope.

means separation from the sclera not only of the tendon, but also of all the subsidiary fibers to the sides of and under the tendon. Such an extensive operation entails surely a decided loss of inward rotation, a permanent weakening or loss of the function of the

FIG. 67.—Bar reading.

muscle, a retrogression of the caruncle and a prominence of the sclera on the nasal side, and possibly proptosis. Parallelism of the visual axes and the correction of the deformity are the immediate apparently satisfactory results. Later, however, the

eyes are likely to diverge, in which event coördination for all distances is lost. We, therefore, recommend *double advancement.* By this operation the previous excess of convergence is neutralized by the acquisition of greater divergence so that by the use of the eyes under proper conditions (hyperopic correction) equilibrium of the lateral muscles is secured and maintained. Inward rotation is not curtailed and no fear need be entertained of a subsequent divergence. At the same time the grasp and control of the whole cone of muscles on the globe is materially increased. By the

FIG. 68.—Remy's diploscope.

operation the images of the two eyes are brought closer together and the fusion power is stimulated to fuse the images. The operation on both muscles should be performed at the same sitting. It may be necessary in cases of the highest grades of esotropia (internus contraction, internus spasm) to later tenotomize one or both interni in addition to the operation of advancement, but this step should be taken only after the lapse of several months, when it will be learned positively that no further increase of divergence may be looked for, as a result of the advancement. For some days, or even some weeks, the operator may be disappointed that

the results are not all that he had hoped for—it is well known that the final degree of divergence is not reached until some weeks after the advancement. This interval should be utilized to aid the development or the increase of the fusion power by exercises of various kinds. Indeed, stereoscopic exercises with the Holmes stereoscope are often of service both before and after operation. Bar reading after the operation (see Fig. 67) is much to be rec-

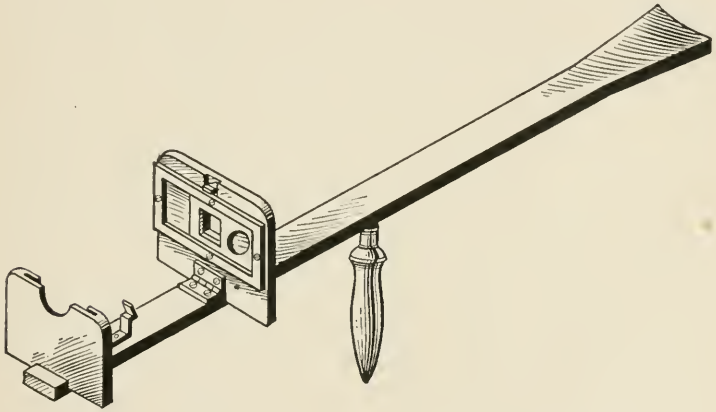


FIG. 69.—Bishop Harman's diaphragm test. See also Fig. 26.

ommended. The diploscope of Remy (Fig. 68) or Harman's diaphragm apparatus (Fig. 69) are both convenient not only to learn whether binocular vision has been secured by the operation, but also as a means of training the fusion sense. These same exercises are of value in monocular esotropia both before and after operation.

EXOTROPIA.

Exotropia may be

1. *Organic*, as a result of destruction of the nucleus or trunk of the 3rd nerve either from traumatism or disease; also as a result of direct traumatism to one of the internal recti.

2. *Partly organic and partly functional*, as in partial loss of vision in one eye from anisometropia or antimetropia, or from injury to the eye; in excessive myopia from elongated eyeballs, in those exotropias the result of too free tenotomies for esotropia in childhood, and in high-grade orbital asymmetry.

3. *Purely functional*, due to the influence on a congenitally weak fusion force (desire for binocular vision) either of some refraction anomaly (usually myopia) or of the natural divergence of the orbital axes in adult life.

Classes 1 and 2 may be binocular, although the deviation is usually confined to one eye.

Class 3, the truly functional exotropias, are almost always binocular, and may be defined as a partial loss of the power of convergence, although the vision of both eyes is alike or nearly alike.

The organic exotropias (Class 1) have been disposed of in a previous chapter; also such of Class 2 as are mainly organic in type. Only those of the second class, depending upon the elongated eyeballs of excessive myopia, or upon asymmetrical orbits, will be mentioned here, and but briefly, the main question under consideration being true functional exotropia.

As has been shown in the previous chapter, esotropia is an active phenomenon growing directly out of overaction of the muscles under the domination of the 3rd nerve.

Exotropia, on the other hand, while due to an anomalous condition of the same group of muscles, is a passive phenomenon, and may in general terms be regarded as a *relaxation* of all the muscles

governed by the 3rd nerve, giving rise to divergence of the visual axes.[1]

It commonly affects both eyes in the earlier stages, in which case it is known as bilateral, binocular or alternating exotropia. In its final phases it is usually confined to one eye, when it is known as monolateral or monocular exotropia.

Etiology.—While the etiology of the affection sometimes seems obscure, the fact seems to stand out that its origin is more or less closely bound up with the presence or development of myopia, which, because of the increased length of the eyeballs and by lessening the demands on accommodation (and hence action on the part of the ciliary branch of the 3rd nerve) lessens in like degree the action of the remaining muscles supplied by the 3rd nerve, and the eyes tend in the direction given them by the normally innervated external rectus and superior oblique muscles— namely, out and down. Thus a deviation of the visual axes not only *out*, but also *below* the horizontal line is likely to be encountered in this, the myopic variety of exotropia.

Should the anomaly be but a functional one up to the time of puberty or soon thereafter, the *deviation tendency* is likely to then become a *true deviation* because of the natural divergence of the orbital axes, which, as has been shown by Weiss, is the feature in the growth of the adolescent and adult orbit. To this latter fact, and to occasional overaction on the part of the superior oblique and external rectus muscles, must be ascribed the exotropia found in the presence of emmetropia and hypermetropia.

It will then be readily seen why exotropia is rarely met with in children (save in high congenital myopes) and why it commonly reveals itself during adolescence or early in adult life. Too, the slow departure of the accommodation in the youthful myope, and in other cases the very gradual increase in divergence of the orbital axes, give to the approach of exotropia an insidious form,

[1] The condition is also known as external squint, divergent squint, strabismus divergens, concomitant or alternating squint or strabismus, and insufficiency of the interni. It is to be hoped that these synonyms will soon give way to the nomenclature here adopted.

while esotropia, on the other hand, is relatively rapid of development. Furthermore, esotropia in the child often becomes esophoria in the youth and may pass through the various degrees of esophoria into muscle-balance when maturity is reached; whereas exotropia in the young or adolescent can only become accentuated as the years wear on. The natural tendency of myopic eyes toward divergence is caused in part by the elongated globes, which must add to the weakness of the adducting muscles in all near work, and in part by the absence of the impulse to accommodation to aid convergence. Very often there is marked irregularity or inequality in vision, and in some cases one eye is so nearly blind that it wanders outward simply in obedience to the tendency impressed upon it by the outward direction of the axis of the orbit. In any case there must co-exist a lack of power of the adductors to render the deformity possible, while refractive conditions play a rôle which has been sufficiently explained.

Symptoms.—The only noteworthy symptoms of exotropia are the deviation, the occasional amblyopia, and the defective vision consequent upon the myopia which usually completes the picture. The deviation itself produces no distress and is often borne for years without any special inconvenience to the patient. Rarely is diplopia present, and then mainly when exophoria is passing over into exotropia. Even under such circumstances the patient is only occasionally conscious of diplopia, and while some patients may be educated into seeing double images, in most instances it is difficult to excite them.

Amblyopia, in the sense of the amblyopia of esotropia, is only rarely a symptom of outward deviation, and if it does occur it is a question as to whether it is of the same origin as the esotropic variety. It has been argued that it grows out of suppression of the image of the deviating eye, habitually resorted to by the patient to avoid the confusion of diplopia, and that if suppression of the images falling upon the retina of the unused eye can be so readily learned as it is by microscopists, ophthalmoscopists, astronomers and others, it seems reasonable to assume that it is re-

sorted to unconsciously, but none the less surely, by exotropics (as also by the victims of longstanding ocular palsies or pareses) to avert the confusion of diplopia. We have no means of learning whether the esotropic infant or child has ever seen well with its deviating eye before that eye deviated, nor whether the complexity of functions that issue in binocular vision have ever been thoroughly acquired and brought into harmonious automatic relations. Exotropia, on the other hand, seldom reveals itself before puberty—long before which time binocular vision has become one of the cardinal functions of the central nervous system. Let it be remembered that the performance of binocular vision calls for harmonious synchronous action on the part of areas in the motor region of the cortex, also on the part of a large portion of the occipital cortex, and lastly on the part of the 2nd, 3rd, 4th, and 6th (namely four out of twelve cranial) nerves. With this proposition in mind, it is not difficult to conceive that many esotropic children have never thoroughly acquired this complex function, and that in such children a moderate degree of hypermetropia suffices to so disturb the act that the brain finally abandons the struggle. Hence we have spoken of a congenitally weak fusion-force. Similarly in exotropia (at least of the myopic variety), when the impulse to accommodation is no longer excited, another disturbing factor has been obtruded on the act of single vision, and the outward deviation of the eye or eyes is the signal of the surrender of the higher centers. However, in exotropia we are dealing with eyes that have enjoyed a high degree of visual acuity, and deviation certainly means diplopia with its sequels unless one eye learns to disregard its images. Necessarily this has naught to do with the amblyopia of those outward deviations which are the result of over-free tenotomies for esotropia in childhood, and which are to be regarded more as pareses of the operated interni than as true exotropias.

It is proper to state that in exotropia there may be ocular symptoms, but these commonly result from the refractive error present, and generally disappear after its correction.

Diagnosis.—The diagnosis of exotropia rests upon much the

same methods as those for esotropia—namely, inspection, the cover-test, with and without prisms, the study of the double images (when they can be induced) with and without prisms, and the tape measure method. Simple inspection will in most cases not only determine the presence of the anomaly, but may also show which is the fixing eye and what the probable degree of deviation. Resort, however, is generally to be had to the cover-test as follows: The patient is seated facing a candle-flame or some similar small bright object 20 or more feet distant. A small piece of card-board is then carried from one eye to the other and if each eye fixes as its fellow is covered, the squint is of the alternating variety. When both eyes are uncovered it will be found that the patient consciously or unconsciously uses either the right or left eye by preference for fixation. We therefore speak of it as the fixing eye. The fellow organ then assumes a varying degree of divergence. Should the deviation be of the monocular or monolateral variety, it will be found, on applying the cover-test, that the deviating or amblyopic eye makes little, if any, movement when the fixing eye is covered. Alternating exotropia may be measured with fair accuracy by using prisms in connection with the cover-test. Having the patient fix on the candle-flame or other object 20 feet distant, a 10 degree prism, base *in*, is brought before either eye, preferably the fixing one. Applying the cover-test, it will be found that the excursion of each eye as the cover is shifted from side to side is much diminished, and by increasing gradually the prism strength, base in, a degree will finally be reached at which both eyes are apparently steady as the cover moves back and forth. Not infrequently it will be found that a vertical deviation co-exists, when it will be necessary to learn the prism needed to arrest the vertical as well as the lateral movement. The prism or prisms required to suppress all movement of the eyes under cover, will be the prism measurement of the deviation; and if in carrying out the test, very high degree prisms are needed, the effect can be divided between the eyes. Those not familiar with the test, will do well to guard against over-correcting with prisms, as this will induce esotropic movement under the cover and thus lead the

surgeon into serious error. The test is purely objective and of great service.[1]

When double images can be excited, we have another valuable test at hand. By coloring the image of the fixing eye with a cobalt glass, the relative position of the images is easily studied, and the degree of the prism that will bring the images into a horizontal or vertical line (or both) readily learned. However, this measure is applicable only to those patients in whom double images can be induced, and who can coöperate mentally with the surgeon in the study of the case; whereas, the cover-test, combined with prisms, renders the surgeon absolutely independent of the patient, and is of signal service in all cases of alternating exotropia irrespective of age or mental condition. The tape measure method is applied in the same manner for measuring exotropia and with the facility as in esotropia. Moreover, as most exotropics are adults the perimeter may be easily employed for the arc measurement of the deviation.

Treatment.—The management of exotropia is optical and operative. At first it would seem that treatment might also be therapeutic; that if atropin serves to interrupt the pathologic process in the early stages of esotropia, pilocarpin or eserin ought, logically, to be of service in the same period in exotropia. Theoretically, this reasoning is correct, but unfortunately it is not borne out by clinical facts. In esotropia, we are dealing with a force that needs curbing, and atropin is the ideal agent. In exotropia, we have to do with functions (accommodation and convergence) that are waning, or with conditions that are the result of anatomic changes (divergence of the orbital axes, and elongation of the globes). The fact is that myotics by stimulating accommodation alone would rather lessen the associated stimulus to convergence, and thus add to the divergence of the eyes. Moreover, it would seem that the instillation of a very weak mydriatic might, by inducing extra stimuli to the ciliary muscle, incite the adductors to action, and hence be of service in the early

[1] The same mistake may be just as easily made in estimating the prism degree of esotropia or right or left hypertropia.

stages of exotropia, although there is no record of such treatment directed to early exotropia.

The optical treatment of exotropia may be affected by correcting lenses alone or in combination with prisms. The greatest care should be observed to learn the exact static refraction in every case, when the following rules are in force:[1]

For non-presbyopes. All myopes under 6 D. to be given a full correction of their optical defect, that the accommodation may be forced into action. It will often be of advantage to assist the accommodation at first with one drop of a one grain to the ounce solution of pilocarpin muriate in each eye three times a day.

Myopes[2] of over 6 D. to be given the fullest possible correction consistent with any degree of comfort in work at the near point. Pilocarpin is also of service in enabling these patients to become accustomed to the new conditions imposed.

Hypermetropes of whatever degree to be given the weakest spherical correction consistent with comfort in working at the near point.

Presbyopes to be given the strongest minus spherical (if myopes), or the weakest plus spherical (if hyperopes) that will enable the patient to conduct work at 13 inches, with pilocarpin (or eserin) again as a synergist.

The object of all the foregoing measures is to induce action of the ciliary muscle in the hope of arousing an associated stimulus to the adductors, and thus lessening the divergence.[3]

If eight to twelve weeks' constant wear of the correcting lenses does not dissipate the exotropia, training of the convergence after the method advised in the chapter on exophoria will be in

[1] Refraction under complete cycloplegia.

[2] In a twenty-year-old young woman who came under our care, wearing -10.00 for distance and -6.00 for near work, the patient was relieved entirelyof her asthenopia and incipient exotropia by two months use of -13.00 D. S. -1.00 degree Cyl. 180 in each eye (her full correction), the employment of pilocarpin as above, and training of the convergence with prisms.

[3] It should be mentioned that this adaptation of the correcting lenses to the exotropia applies to their spherical components only. Any cylindrical element necessary should be prescribed in its full static strength.

order, but this is feasible only in the lowest grades of exotropia where binocular vision is still possible. Anything over 15 degrees falls out of this class. As a last optical resort, streoscopic training of the adductors may be tried after the method of Landolt, and some few patients will respond to this method of educating the fusion impulse after all other methods have failed. Finally, if after optical correction, the deviation be less than 15 degrees, and does not respond to convergence training, or if it is over 15 degrees, operation is the final resource. Inasmuch as we are dealing with a passive phenomenon, shortening of the relaxed muscles is indicated, rather than tenotomy of their antagonists, hence advancement is preferred to tenotomy. In monolateral exotropia, the deviating eye should always be approached first. If vertical deviation co-exist, it should be remedied before proceeding to advancement of the internus of the squinting eye, and if these two steps leave a residual deviation, tenotomy of the externus of the squinting eye may be performed. Any deviation then remaining must be met by advancement of the internus of the sound eye. Rarely will it be necessary to go on to tenotomy of the externus of this eye.

In binocular exotropia, the muscles to be attached are taken up in the following order: Any vertical imbalance must be first corrected; efforts to restore the lateral balance are then made by advancement of the internus of the eye which does not fix; or, if fixation be indifferent, by advancement of the internus of that eye which presents the poorer corrected vision. If this prove insufficient, advancement of the internus of the fellow-eye becomes necessary, and if external deviation persists despite all these procedures, tenotomy of either or even both externi is in order. These latter measures, however, will be required only in the most aggravated exotropias. In those few cases of pure divergence excess with fairly well preserved convergence faculty (the so-called neuropathic divergence), which usually are associated with hypermetropic states of refraction, tenotomy of both externi may be done with much confidence.

As a rule, exotropes who have once possessed the faculty of bin-

ocular vision are much more easily restored to its privileges than young esotropes who, in all probability, have never enjoyed good binocular vision. Hence, the prognosis of the operative treatment of exotropia is favorable, although the length of time necessary to bring the visual axes into coördination extends in some cases over several months. In those exotropias which are the result of too free tenotomies for esotropia in childhood, the divergence is usually monocular and is very wide, frequently reaching 55 or 60 degrees. In such cases it becomes necessary to do a wide resection and advancement of the internus with its overlying conjunctiva and surrounding capsule combined with tenotomy of the externus of the same eye. It is well to secure 5 to 10 degrees of over-effect in such cases, as a portion of the effect gained by operation is usually lost within two to three months after the operation.

HYPERTROPIA.

Hypertropia, or true vertical deviation of one visual axis above the other, is not often encountered alone, but is usually associated with either esotropia or exotropia Admitting the relative infrequency of pure uncomplicated hypertropia, one cannot but be struck with the meagerness of references to this anomaly in the most recent as well as in the older text-books on ophthalmology; nor does recent ophthalmic literature throw much light on the subject. And the foregoing fact is even more striking when it is remembered that the same causes which underlie the *functional* or latent vertical imbalances (hyper*phorias*) lead directly up to and are as distinctly causative of, the actual vertical deviations or hypertropias.

Etiology.—The ciliary overaction which in hypermetropic eyes often gives rise to a temporary esophoria or hyperphoria, may, under certain conditions, carry the anomaly from the latent or heterophoric class over into the actual or heterotropic phase; in other words, long perverted physiology finally issues in pathology. Just so long as the patient's fusion power asserts itself, just that long will the patient preserve binocular vision and in all likelihood present a train of ocular or reflex symptoms as the result of the extra output of nerve-force necessary to maintain fusion. During this stage *deviation tendencies* only can be elicited —in other words, we are dealing with heterophoria; and many patients will carry a defect of this kind throughout life. But it frequently happens in childhood or adolescence that the supply of nerve-force necessary to fusion is not equal to the demand, and the muscular system of one or the other eye gives up the unequal struggle and latent tendencies become manifest, or heterophoria (functional turning) advances now to heterotropia, or actual turning. This variety results in the vast majority of cases either from uncorrected errors of refraction or

from the change in the dimensions and direction of the orbital axes common to the time of puberty. Hence, hypertropia is similar in origin to esotropia and in many respects to exotropia, with either one of which it is therefore nearly always associated, and upward is frequently associated with inward deviation as seen in Fig. 69.

In hypertropia either the right or the left visual line may be habitually higher than the other (right and left constant hypertropia respectively); or each visual line may be alternately higher than the other—alternating hypertropia. Vertical squint is further classified as strabismus sursumvergens when the lower eye is the one that habitually fixes and strabismus deorsumvergens when the upper is the fixing eye. The preceding statements made in particular reference to the etiology of regular hypertropia (a deviation of one visual axis above the other) seem to us none the less applicable to a certain remarkable class of cases, mentioned by Stevens,[1] in which *both* visual axes deviate either above or below the horizontal plane when the head and eyes are in the primary position. Deviation of both axes upward is termed anatropia. That is, in anatropia, the right eye deviates up behind the screen or cover when the left is fixing, the left eye moving up also (instead of down as in hypertropia) the instant the cover is carried to that eye and the right eye is permitted to fix. Katatropia is present when with the same cover-test the excursion is down in both eyes.

It is our belief that anatropia is an overaction phenomenon and that in all probability it is closely related to esotropia, a similar phenomenon. The excessive impulse which in hypermetropia is sent to the ciliary muscle, affects in like manner the remaining muscles supplied by the third nerve, in consequence of which the eyes tend in the direction resulting from the combined action of those muscles, namely, up and in. If in esotropia the cover-test be used with the patient fixing on some point 20 or more feet distant, it will be found that both eyes deviate not only inward, but also upward, as the cover is shifted from one to the

[1] *Annals d'Oculistique,* April, 1895.

other eye (Fig. 70). On the other hand, the absence of impulse to the ciliary muscle of myopes (who commonly use little if any accommodation) carries with it a very much diminished impulse to the other muscles governed by the 3rd nerve, and their resultant inaction permits the eyes to be deviated in the direction resulting from the combined action of the remaining ocular muscles (the external rectus and superior oblique), namely, down and out. The cover-test in such a case, provided binocular vision can be

Fig. 70.—Upward and inward deviation of right eye. Such upward inward deviation of the right eye is sometimes seen with congenital paresis or palsy of the left superior rectus with spasm of the inferior oblique of the right eye (the associated muscle) especially if the eyes were carried toward the left.

maintained, will show that both eyes deviate both outward and downward under the cover or screen as it is carried from one to the other eye.

The organic hypertropias (generally paralytic), are seen principally in grave cerebrospinal disorders and have been considered at some length in the chapter devoted to palsies. These forms of hypertropia must not be permitted to obscure our ideas about true right and left hypertropia and strabismus sursumvergens and deorsumvergens, whose origin is closely bound up with anomalies of refraction and with the shape and development of the orbits.

The following case is in point. M. B., a twelve-year-old girl, came with a history of a peculiar appearance about her right eye since her third year. No history that is worth while obtained. Inspection shows rather marked facial asymmetry. The right orbit measured 3 mm. less in the vertical and transverse diameters than the left, also the right eye was 3 mm. deeper in the orbit

than the left. There was marked upward deviation of the right eye that measured 16 degrees constantly. At times esotropia was present, at other times it was not. It varied from 15 degrees to 20 degrees. With the tropometer the rotations of both eyes were as follows: R. E. Up 46 degrees. Down 57 degrees. In 45 degrees. Out 40 degrees. L. E. Up 38 degrees. Down 48 degrees. In 60 degrees. Out 35 degrees.

She had never worn glasses. Her refractive status was

R. + 6.00 sph. + 1.00 cyl. 90 degrees = 5/60
L. + .50 sph. + .37 cyl. 105 degrees = 5/5

which was ordered for her when her esotropia disappeared entirely, the vertical element remaining. After 6 months use of the glasses, the vertical deviation remained at 15 degrees, when tenotomy of the right superior rectus was done with most gratifying results. Of course she continues to wear her glasses.

Symptoms.—The symptoms of the earliest stages of hypertropia are largely those of hyperphoria. However, when the weak depressors or elevators have abandoned their fruitless task, the eye to which they belong will deviate in the direction of action of the stronger muscles, binocular vision will be lost and along with it will most likely disappear much of the patient's ocular and reflex discomfort. The resulting deviation which is the main objective symptom, is easy of recognition in most cases, especially with the cover-test at 20 feet. The cardinal subjective symptom is the amblyopia of the hypertropic eye. In recent or low-grade vertical deviations, especially if they are alternating, even corrected vision will be compromised to some extent in both eyes, although more decidedly so in the affected eye. In old cases the fixing eye will nearly always present a close approach to normal vision (corrected if necessary) while the vision of the other eye is often lowered to a remarkable degree.

The mooted question as to whether the amblyopia produces the deviation or the deviation the amblyopia is discussed under the chapters on Esotropia and Exotropia. Aside from the deviation and the amblyopia, there is no symptom worthy of note unless

it be the peculiar "wall-eyed" appearance of such patients, especially if the hypertropia be alternating, giving to the patient the well-known hyperphoric or hypertropic "stare." Partial ptosis not ascribable to other cause is regarded by some authorities as symptomatic of hypertropia.

Diagnosis.—The presence of this anomaly is easily detected, and in most cases its character and the eye to which it belongs can be determined almost at a glance. In the alternating variety resort must frequently be had to the cover-test to learn which eye fixes by preference. This test will be productive of the best results if carried out in a half-darkened room, using a small gas-flame or some other luminous point as the fixation object. By interposing a weak prism before the higher eye with the apex of the prism placed in the direction in which the eye deviates, and increasing it in strength until the eyes no longer move visibly when the cover is carried from one to the other eye, the deviation may be measured with a reasonable degree of accuracy. This method is applicable also to those cases in which the patient cannot be made conscious of diplopia, and it enjoys the additional advantage of being an objective method, thus rendering the surgeon entirely independent of the patient's testimony. In cases that are, or can be made, conscious of diplopia, the use of the cobalt

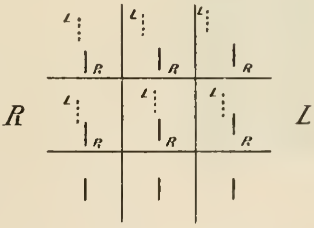


Fig. 71.

glass before the sound eye is of great value, and all that will be necessary is to find a prism that brings the two images to a horizontal level. The Maddox rod is employed by some surgeons in the diagnosis of hypertropia, but its use is largely restricted to the estimation of deviation tendencies rather than to actual deviations. In all cases of hypertropia, search should be made in the extreme upper periphery of the binocular fixation field for diplopia which if found generally implies paresis of one or the other superior rectus. Thus J. C., sixteen, male, diphtheria six years ago following which the left eye "took on a peculiar appearance."

Clinically right hypertropia of 20 degrees seemed the proper diagnosis but search for diplopia revealed it most marked in the left upper field with the left image higher (Fig. 71). The tropometric rotations were R. E. up 35 degrees, down 50 degrees, nasal 55 degrees, temporal 50 degrees. L. E., up 15 degrees, down 60 degrees, nasal 50 degrees, temporal 45 degrees. Paresis of the left superior rectus was thus plainly shown with a diagnosis of paretic left *hypo*tropia.

Advancement of the left superior rectus and tenotomy of the left inferior rectus at the same sitting gave him approximate vertical balance, (left hyperphoria of 2 degrees).

Treatment.—The treatment of the varieties of hypertropia under consideration is orthoptic and operative.

, By orthoptic treatment is meant the persistent use of a full cycloplegic correction of any ametropia that may be present. The effect of such lenses will frequently be much enhanced by continuing the effect of the atropin or other cycloplegic for from eight to twelve weeks, in the hope of thus re-establishing proper relations between accommodation and convergence.

FIG. 72.—Typical left hypertropia.

In young subjects (under ten or twelve years) these measures will frequently lessen the deviation and sometimes dissipate it altogether. Oftener, however, and in subjects over twelve years of age in particular, a considerable deviation remains, which must be met by some operative procedure (Fig. 72). The laws governing operative interference in heterophoria are here in force. Overaction hypertropias, mainly those found with esotropia (including anatropia) demand tenotomy of the overacting muscle or muscles of the higher eye to be combined in some cases with tenotomy of the associated muscle or muscles of the sound eye. Underaction hypertropias (usually hyperexotropias or exceptionally katatropias) will be best met by advancement or muscle-shortening of the faulty muscle or muscles of the lower eye, and, where this is insufficient, by a similar procedure on the muscle at fault in the

fellow eye. It is to be remembered that division of the superior rectus acts by association on the levator palpebræ superioris, and is followed, not only by depression of the cornea, but also by lifting of the upper lid, in consequence of which a considerable amount of sclera will be exposed above the cornea. However, this fact may be utilized in cases of partial ptosis, both to aid the elevator of the lid as well as the depressor of the cornea. It is further to be borne in mind that operations on hypertropes over twelve years of age are frequently cosmetic operations pure and simple; and, notwithstanding Stevens' claim that restoration of binocular vision should be the ultimate aim in these cases, such termination of them is rare and must be viewed only as a most fortunate circumstance.

Finally, operative treatment is contra-indicated in hypertropia of variable degree; or of recent origin and progressive; in paretic cases of central nervous origin; and in rheumatic or gouty individuals well on toward fifty years of age.

PART IV.

OPERATIONS.

ANESTHESIA.

In heterophoria, the success of the operation depending on a partial or accurately measured tenotomy, local anesthesia is essential. Indeed, a precise operation cannot be performed unless the patient is conscious and can advise during the procedure of the effect obtained by the various steps.

In heterotropia (or true strabismus) general anesthesia will be frequently necessary. Operations for convergent strabismus are oftenest done on children under twelve years of age and there are few children who are stoical enough to bear the procedure under local anesthesia whether the operation be tenotomy or advancement. This is always a disadvantage in that the normal coördinations are set aside by the general anesthetic and the eyes drift outward and upward into the position normally assumed during sleep. Under these circumstances one can form no judgment as to the amount of surgical correction necessary, and will have to be guided entirely by previous experiences as to how much to tenotomize, how much tissue to remove, or how far to advance a tendon in a given case.

In adults many operations (even advancement) may be done under local anesthesia, especially if conjunctival and subconjunctival anesthesia are combined.

Ordinary conjunctival anesthesia is obtained by instilling one or two drops of whatever anesthetic may be used every three minutes for five applications. This will suffice in all tenotomies done without the strabismus hook and in some done with the hook. In the latter method, however, the patient often complains of much discomfort when the muscle is lifted on the hook. Indeed it is the most trying period in the operation. We have found that the use of an anesthetic subconjunctivally after con-

junctival anesthesia has been produced contributes greatly to the patient's comfort. The following solution is used:

4 per cent. sol. cocaine muriate,	1 fluiddram.
Normal saline solution,	1 fluiddram.
Solution adrenalin, 1 : 2000,	1 fluiddram.

of which 5 to 8 minims are injected with a hypodermic needle under the conjunctiva over the muscle or muscles to be attacked. During the ten minutes wait for this solution to act, gentle continuous pressure is made on the eye wth a gauze pad to so diffuse the introduced liquid that it will affect the widest possible area and also to disturb as little as possible the natural topography of the parts. The operation may then be approached with fullest confidence and with a comparatively bloodless field because of the adrenalin preparation. In many adults, if they have any pluck at all, advancements may be done almost painlessly by this method. The advantage of having the patient conscious with the normal innervation to the eye-muscles during such an operation cannot be overestimated. Without subconjunctival anesthesia, relatively few advancements can be performed even on the most phlegmatic adults. Naturally other anesthetics such as holocain hydrochloride, novocain, alypin, stovaine, or beta eucain may be employed, but some adrenalin preparation should be used with them whether the anesthesia be conjunctival or subconjunctival. We have used beta eucain with much satisfaction. It cannot be too much emphasized however, that in children under twelve to thirteen general anesthesia will almost invariably be required.

ANTISEPSIS. ASEPSIS AND INSTRUMENTS.

The operator's hands and nails should be scrubbed with warm water and soap until all mechanical impediments to the action of the chemical agent to be used immediately afterward are completely removed. The hands are then dipped in a solution of bichlorid, biniodid, or cyanid, of mercury, 1 to 3000. The skin of the patient's face in the neighborhood of the eye is scrubbed

with warm water and soap, then douched with normal salt solution, and washed finally with 1:6000 mercurial solution. In this manipulation, particular attention must be paid to thorough cleansing of the eyebrows and lashes. The conjunctival sac and the conjunctival surfaces of the lids are flushed with physiologic salt or saturated boric-acid soluton, and fresh cocain solution is applied. The instruments are first placed in boiling water and then in alcohol, where they are allowed to remain until used. This simple method of treating the instruments is sufficiently germicidal for all operations where the field is not unusually septic.

TENOTOMY.

The object of tenotomy is two-fold: By altering the tendinous attachment of a muscle to change its mechanical relations to the globe and to the other muscles, and by thus lessening the power of the muscle to so influence the distal response to innervation, that equilibrium and coördination shall be inaugurated or reestablished; second, to develop or restore symmetric and corresponding nerve-excitation. We are concerned, first, with the muscle or muscles at fault, and, second, with the degree of deviation—*i. e.*, whether there shall be partial or complete muscle-division, and whether it shall apply to one or both eyes.

In heterophoria, unless there is good ground for believing that one muscle is abnormal either in structure or insertion, the operation should in all cases be divided between the two muscles, relieving an equal amount of tension in each. In heterotropia (esotropia for instance) the evils resulting from extensive tenotomy in one eye for high grade deviation are limited movement nasalward, diplopia in the periphery of the field, cicatrization of the conjunctiva and capsule at the site of the wound, retraction of the caruncle and protrusion of the eyeball. In monolateral strabismus with high grade amblyopia it is our practice to do a moderate tenotomy of the internus and extensive advancement of the externus of the deviating eye. Later, if necessary, advancement may be done on the externus of the sound eye. The same

principles obtain in exotropia, save that the muscles to be attacked are of course reversed. In esotropia or exotropia without well marked amblyopia, advancement of both externi or interni (as the case may be) is the operative procedure of election.

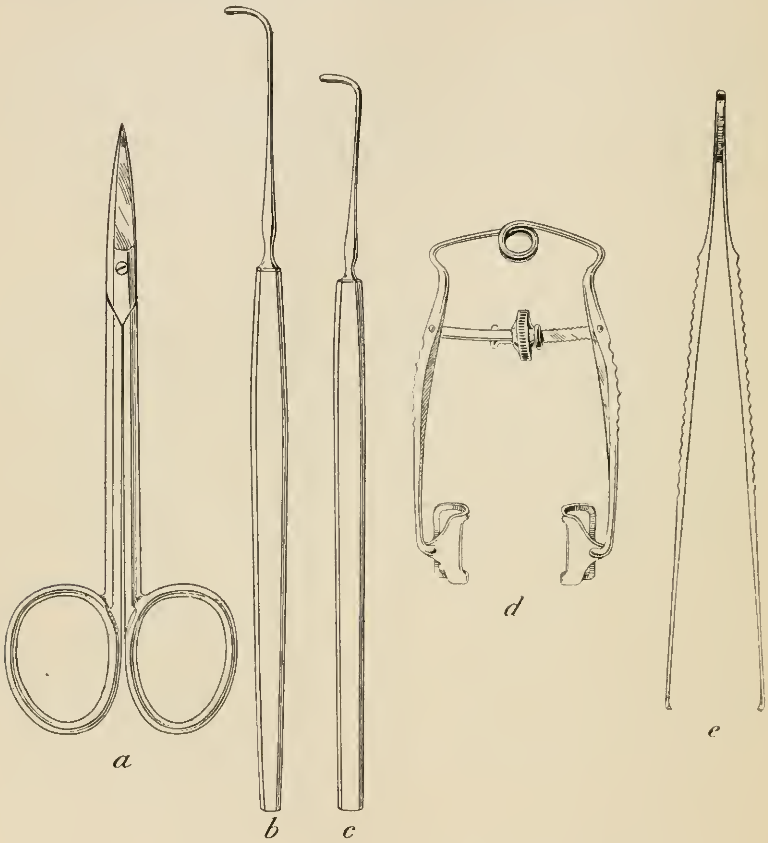


FIG. 73.—Instruments used in tenotomy. *a*, scissors; *b* and *c*, tenotomy hooks; *d*, speculum; *e*, conjunctival forceps.

After insertion of the speculum or separation of the lids by an assistant (lid elevator held in one hand and the lower lid depressed by a finger of the other hand) the conjunctiva and the capsule lying immediately over the insertion of the tendon into the sclera are firmly grasped by the single tooth conjunctival forceps.

Considerable pressure with the forceps at right angles to the ball is necessary to ensure the embrace of the capsule with the mucous membrane. The advantage of securing both can be appreciated in the next step, the incision. If both structures can be divided and the tendon exposed at its insertion by one snip of the scissors, this part of the operation is considerably simplified. Two slight difficulties present themselves in case the conjunctiva alone has been divided; the smooth surface of the capsule uncovered by mucous membrane and made tense by outward rotation of the cornea, is slippery and hard to grasp; and again, unless one is cautious he is apt to include the tendon in the grasp of the forceps and to divide it unintentionally. The incision should not exceed the width of the tendon; if the desired result is not obtained it may be widened at the close of the operation; if it be too narrow, the necessary manipulations are hindered (Fig. 74). A few operators prefer to make the direction of the incision horizontal, parallel and close to the lower border of the tendon, but the common practice, and the better one, is to make it vertical in order that it may have the greater influence in the regulation of the final result. Having exposed the tendon at its attachment, a small hook is passed under its entire width from above or below, the distal end of the hook coming into view at the upper or lower extremity of the incision, using the scissors to snip away any capsular folds that the hook is likely to push before it. The tendon may now be divided to any extent desired (see Fig. 74). The method recommended for the correction of heterophoria is to incise first the middle fibers and then if necessary divide toward the marginal, according to the degree of effect previously determined, gauging the progress toward equilibrium by frequent interruptions of the operations for exam-

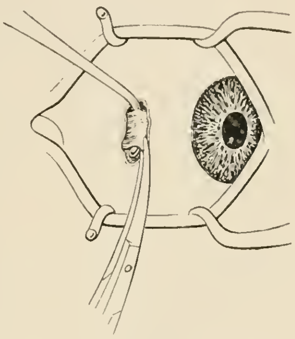


Fig. 74.—Showing size of vertical conjunctival incision also tendon about to be divided.

ination by the tests above described. In heterotropia, the division of the operation into these stages is less important since the tendon is to be detached in almost its entire width. If section of the tendon alone will not restore equilibrium, the conjunctival wound can be enlarged and the fibers radiating from the upper and lower margins of the tendon and from the capsule of Tenon can also be severed, but care must be exercised that the evil effects of too extensive division of the capsular attachments already alluded to shall be avoided. The value in prism degrees, of section of the tendon varies in different cases from 2 to 10 degrees or more. In graduated tenotomy—*i. e.*, division of a few fibers in the center of the tendon, or of both margins the effect is decidedly limited (see Fig. 53); when the whole tendon is severed, without additional incisions of the conjunctiva or capsule, the effect would average about 8 degrees. In proportion to the tissues divided and the extent of the wound the effect may vary from slight weakening of the muscle to a practical paralysis. Union of the tendon to the sclera may take place so far back that the muscle loses all power of rotating the cornea, or, indeed, it may always remain detached. Perhaps our figures as to the effect secured may seem small, but they are the result of an experience numbering several hundred tenotomies and they are, we believe, as correct as general averages usually are.

Tenotomy or advancement of the superior or inferior recti may be performed after the same principles as the same operations on the lateral muscles. The operator should bear in mind the relative less power of the vertical than the horizontal and consequently confine his operations within narrower limits. Since the defect requiring operation is proportionately less (almost one-third), the effect of complete tenotomy or extensive advancement or resection, is almost prohibitive of subsequent binocular fixation in all portions of the field.

Operations on the oblique muscles are rarely indicated. The effect of such operations may be more accurately obtained by operations on a lateral combined with an operation on a vertical muscle.

The after-treatment is simple. Cold applications for a few

hours and frequent instillations of saturated boric-acid solution are all that are necessary in the majority of cases. A bandage is seldom used; on the contrary, it may be positively harmful in that, in the exclusion of binocular fixation, the important factor to success, namely, the unconscious effort at fusion, is prohibited. If immediately, or a day or two after operation, the defect is found to have been over-corrected, a suture to either close the conjunctival wound or a deeper one that shall include the capsule and the severed muscle may be inserted. It is the practice of some operators to introduce the suture before cutting the muscle, so that an over-defect may be instantly neutralized; but this is, we believe, an unnecessary precaution for a skilful operator.

Accidents.—It occasionally happens, through an anomalous distribution of the large vessels, that one of them is torn or cut, producing immoderate hemorrhage. This is especially true of the vertical muscles. In this unfortunate event the operation should be suspended and a pressure bandage applied. A second accident is the unexpected perforation of the sclera by the scissors while cutting the tendon, which must be ascribed to using too sharp-pointed scissors or to cutting with the ends of probe-pointed ones at right angles to the tendon (instead of on a line with the muscle), with undue force. The operation must be at once discontinued, the conjunctiva sutured over the perforation, and a pressure bandage applied.

Healing.—After the usual operation, the tendon re-attaches to the sclera in from three to four days. If the wound should become infected, tenonitis, orbital phlegmon, ulceration of the sclera or cornea, or panophthalmitis may ensue. Tenotomy has been known to be the exciting cause, also, of detachment of the retina, essential phthisis, and hemorrhage fatal to the sight and to the integrity of the ball.

Disappointment as to Result.—A perfectly performed tenotomy will sometimes fail to favorably affect the deviation for which it was undertaken, in which event it is not unlikely that the failure can be ascribed to some anomalous arrangement of the check ligaments which accompany the recti muscles. (See Figs. 4, 5, and 6.)

ADVANCEMENT OR RESECTION.

As has been said in the chapter on Tenotomy, local anesthesia (by combined conjunctival and subconjunctival anesthesia) should be employed whenever feasible in all advancement or resection operations. In children under fourteen, general anesthesia is imperative and nothing but experience will guide

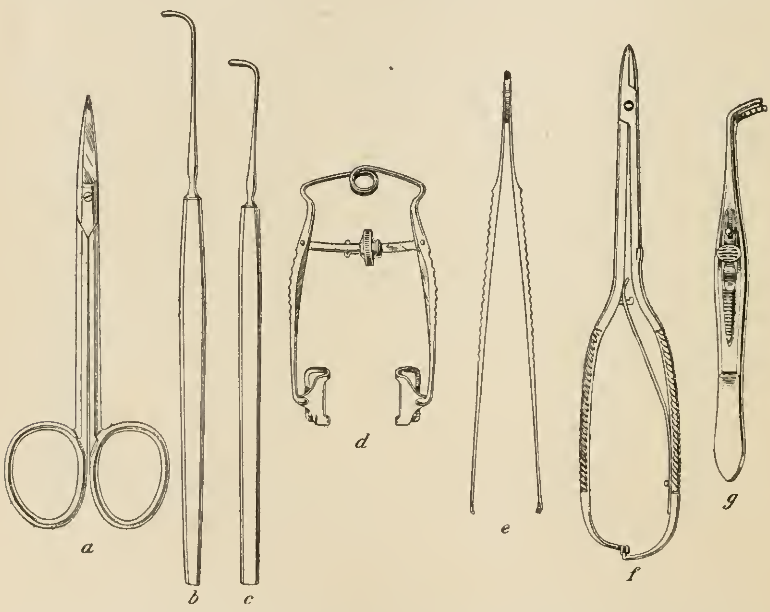


FIG. 75.—Instruments used in advancement or resection operations. *a*, scissors; *b* and *c*, strabismus hooks; *d*, speculum; *e*, conjunctival forceps; *f*, needle holder; *g*, Prince's clamp or advancement forceps.

the operator under these circumstances in the amount of tissue to be excised or the distance a tendon is to be advanced.

Numerous operations for increasing the efficiency of any one muscle have been advocated. Their number points to the inadequacy of any single procedure. The object may be accom-

plished either by advancement of a muscle or tendon, or by resection—or cutting out of a piece of a tendon or muscle. Naturally each of these methods has its supporters. It seems to us wise to omit the description of the great majority and to confine ourselves to those which have proved most efficient in our hands. Fig. 75 shows the necessary instruments.

The principal objection to be urged against most advancement operations is, that the sutures by which the advanced tendon is attached to the conjunctiva frequently tear out, allowing the tendon to recede to a point farther back on the ball than that to which it was formerly attached, and the original deviation is thus aggravated. The advantage of resection or shortening a muscle is that there is no danger of increasing the deformity since the tendon, at its attachment to the sclera, is not disturbed and the shortening process is confined to the muscle and tendon.

When small effects are desired our preference is for an operation that is a modification of the tendon folding of LaGleyze. Excepting instances of marked deformity, in which the purpose of operation is mainly cosmetic, this measure affords satisfactory results in many cases. Its effect can be accurately gauged; it is absolutely without danger of increasing the original deformity; it is attended with but moderate traumatism, and the healing is, as a rule, uneventful and rapid.

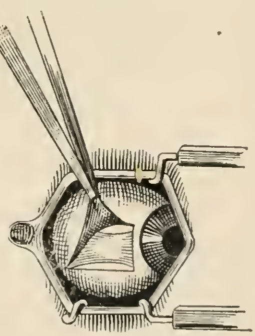


Fig. 76.—Exposure of the muscle.

First Step.—After anesthetization the conjunctiva is grasped with the forceps over the inferior corner of the tendon-insertion and divided vertically 4 to 5 mm., and (starting at the same point) horizontally along the lower border of the muscle as far back as is necessary to given a roomy field for operation.

The subconjunctival tissue and the capsule of Tenon are similarly treated, the structures thus far divided forming a flap, which is turned upward (Fig. 76).

The Second Step.—The muscle is now brought plainly into view by the strabismus hook and freed from all adhesions, when a black silk thread, armed at each end with a fine curved needle, is introduced into the substance of the muscle from 6 to 8 mm. back of its insertion, one needle passing through the upper border of the muscle from its conjunctival to its scleral surface, the other through the lower border in like manner, and the thread drawn taut over those fibers included between the two points of entrance of the needles.

Third Step.—The needle belonging to the upper portion of the muscle suture is now passed under the tendon at the *upper* border

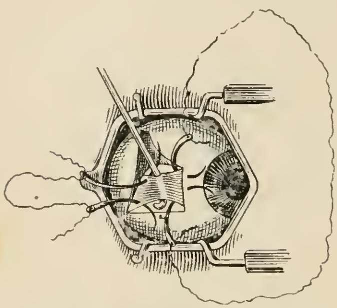


FIG. 77.—Introduction of sutures.

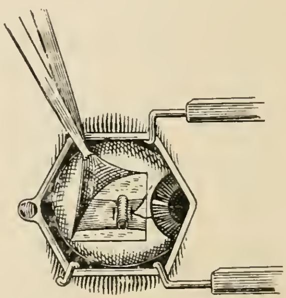


FIG. 78.—Tying of sutures, producing knuckle in muscle.

of its insertion and brought out near the center of the conjunctival surface of the attachment; the second needle, belonging to the lower portion of the muscle suture, is introduced in exactly the same manner under the *lower* border of the tendon-insertion and brought out on the conjunctival surface just below the first needle (Fig. 77). An assistant now grasps the muscle back of the point where the needles were first introduced and brings the muscle forward, when the suture is drawn tight and tied, by this measure advancing the whole muscle nearly as far as the tendinous attachment and producing a fold or hump in the muscle (Fig. 78). The summit of this muscular fold or hump is then cut away, leaving the upper and lower borders intact and the two raw

surfaces in juxtaposition (Fig. 79). The suture is cut off so that its ends may protrude a short distance through the conjunctival wound, which is now brought together by two sutures (Fig. 80) or more sutures in each side.

Pain in the healing may be largely prevented by the constant application of ice-water compresses and frequent instillations of a solution of boric acid and cocain. The conjunctival threads may be removed in a few days, and the thread in the muscle should be allowed to remain as long as it causes no irritation. Any difficulty that might be experienced in the removal of this deep thread can be forestalled by leaving long ends to it, so that it may be readily brought away by the forceps.

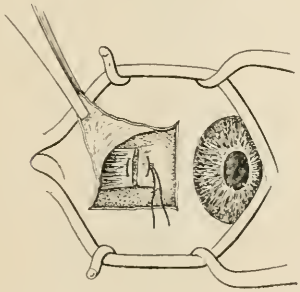


FIG. 79.—Knuckle cut off leaving ends approximated.

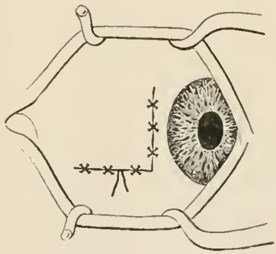


FIG. 80.—Finished effect after tying of conjunctival sutures.

Capsular Advancement.—Another method of correcting the smaller deviations is to advance the capsule of Tenon, as recommended by de Wecker.[1] It consists essentially in incising the conjunctiva freely in the vertical meridian about 4 to 6 mm. from the cornea; then, grasping the capsule over the insertion of the muscle at fault, sutures are made to take firm hold on the capsule and the free ends are carried under the conjunctiva, one above and the other below, as far as the middle of the cornea. These sutures are then tied with careful attention to the traction they produce on the advanced capsule, so that the tension will be equal above and below. This operation, known as capsular advancement,

[1] De Wecker and Masselon, *Man. d'Ophthal.*, Vol. II, p. 778.

is indicated only in the smallest degrees of heterophoria, and is of no value in well-established squint.

For larger and cosmetic effects in the grosser deviations some such operation as Wootten's or Worth's is our preference. Worth proceeds by incising the conjunctiva and capsule vertically (Fig. 81, 1), and then bringing the muscle plainly into view on the strabismus hook (Fig. 81, 2), when it is freed from all attachment for a distance of almost 8 to 10 mm. from its insertion. At this point Prince's advancement forceps are introduced (Fig. 81, 3) and made to clamp between its blades the conjunctiva, capsule and muscle, the upper blade of the forceps resting on the external surface of the conjunctiva, the lower one on the sclera. (It will thus be seen that in this operation a resection is done of all the tissues including the conjunctiva, capsule and muscle). The sutures are now introduced. Beginning within and near the upper border of the muscle, a needle is carried from without inward 3 to 4 mm., back of the blade of the forceps through all the structures (Fig. 81, 4). On emerging on the scleral side the needle is immediately reversed in the needle holder and carried back parallel to the way it was brought forward, but just above the upper border of the muscle, coming out through the capsule and conjunctiva (but not including the muscle (Fig. 81, 5). The suture is then tied down on the external surface of the conjunctiva and thus secures in its grasp a few fibers of the muscle. A separate suture armed again with one needle is introduced similarly at the lower border of the muscle, being carried in and out in the identical manner and then tied (Fig. 81, 6). The tendon is now severed from its main attachment and all fine fibrous subsidiary attachments dissected away. Turning back the muscle and its accompanying structures on the Prince's forceps, the needle belonging to the upper suture is again passed through all the structures, being introduced 1 mm. directly back of the knot previously made and the suture is carried forward and anchored firmly at the original site of insertion of the tendon, care being taken to get a good bite in the episcleral tissues with the needle before the suture is drawn

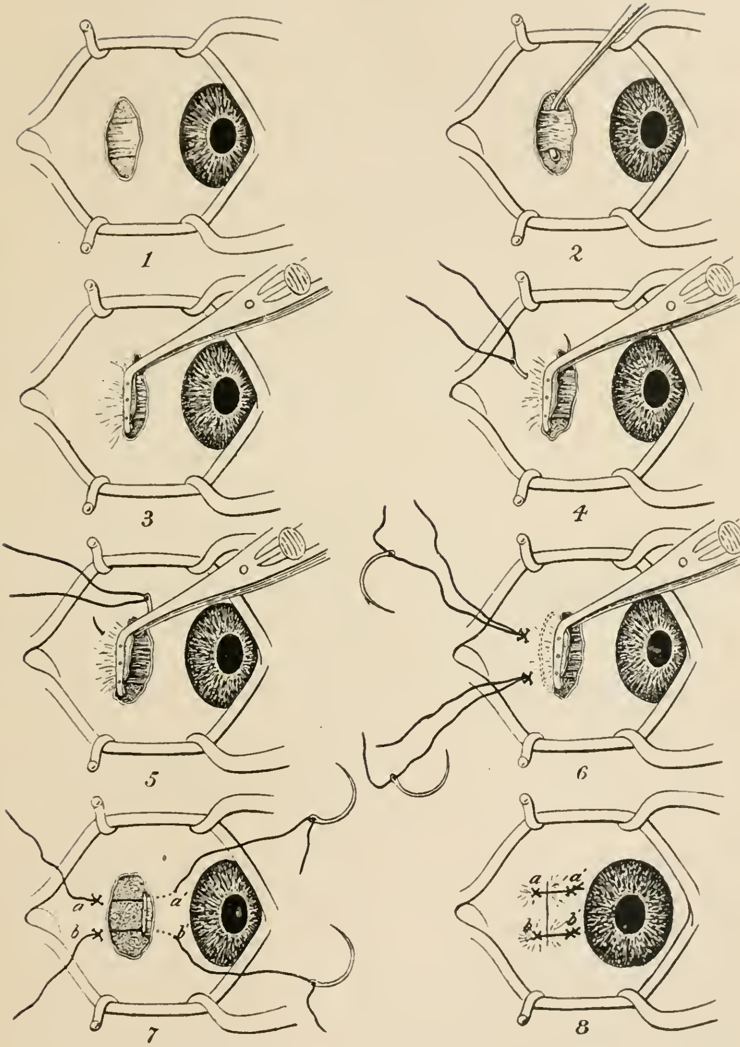


FIG. 81.—Diagrammatic representation of Worth's operation.

through. The same step is now taken with the lower suture (The object of bunching up the muscular fibers in each suture as first tied is to afford the traction suture inserted behind it something definite to pull on without the danger of pulling out through the muscular fibers, a post-operative accident that occurs none too infrequently). The next step is to excise the portion of the tissues held in the grasp of the Prince's forceps by cutting with the scissors between the forceps and the knots adjoining the traction sutures when quite a portion of the sclera is exposed (Fig. 81, 7). The final step of the operation is to tie firmly together the two ends of the suture *aa′* and then similarly tie down firmly the two ends of the suture *bb′*, which should result in good approximation of the cut end of the muscle to the original site of insertion of the tendon (Fig. 81, 8). If there is any gaping at the center of the approximated structures it is good practice to introduce a third suture to facilitate smooth healing and afford additional support to the previously introduced traction sutures. It is difficult to say in arbitrary terms just how much tissue is to be excised, but under ordinary circumstances removal of a 3 mm. wide strip produces a satisfactory result. When it is decided to do tenotomy and resection at the same time on one eye, it is best to perform the tenotomy just before the final tying is done with the advancing sutures in the resection operation. This enables the surgeon to get the fullest effect out of the resection operation. One marked advantage of the operation just described is that all the knots are tied in the external surface of the conjunctiva, so that when they are to be released and removed no difficulty whatever is experienced.

When advancement or resection operations are done on one eye only (*under local anesthesia*), the patient may sometimes be allowed to go home immediately afterward if the operation has been done under local anesthesia. He should be cautioned to remain perfectly quiet at home for two days with the eye or eyes bandaged. Any advancement operations done under general anesthesia whether upon one or both eyes require that the patient should

remain in bed for forty-eight hours with one or both eyes bandaged (as the case may be) throughout that time. If at the end of forty-eight hours the position of the eye is satisfactory to the surgeon the bandage should be reapplied for twenty-four hours longer—but if the effect seems too great the bandage may be removed in the hope that the antogonistic muscle may help to stretch the tissues during healing.

Wootton's operation is somewhat similar to the one just described, differing mainly in the method of introducing the sutures which will be seen at a glance in referring to Fig. 82. There is the possibility, however, that the sutures may pull forward and out through the fibers of the muscle in this operation as in the modification of the LaGleyze operation above described. Valude, Sydney Stephenson, Verhoeff, Briggs, Reese, Vard Hulen and many others have offered advancement or resection operations that differ from the above operations in one or another detail, but we have found the one fully described of the greatest service and to it we pin our faith.

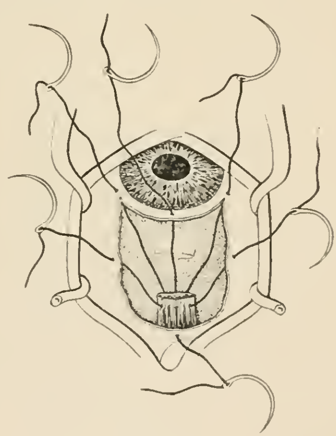


Fig. 82.

After-Treatment.—After the operation is completed 20 per cent. argyrol solution should be instilled and both eyes bandaged, with the object of preventing infection inducing rapid healing, and, by removing any stimulus to movements of the eye, prevent tearing out of the threads. The inclusion of the unoperated eye in the bandage is important even though only one eye has been operated upon, for by this means only will the eyes be kept still. Twenty-four or forty-eight hours later the bandage may be removed, the face washed with bichloride solution, the crusts on the lashes dissolved and the conjunctival sac washed out with boric acid solution. Instillation of a few drops of argyrol (20 per cent.

solution) is recommended before the bandage is reapplied. In monocular operation the bandage may be omitted after forty-eight hours; in binocular it should be continued for twenty-four hours longer.

The postoperative reaction usually subsides in from one to two weeks, although the eyes may remain congested for several weeks. Speaking generally, the sutures may be removed (under cocaine anesthesia) at the end of a fortnight.

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